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HUMAN HEALTH BASELINE RISK ASSESSMENT
Beede Waste Oil/Cash Energy Site
Plaistow, New Hampshire

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ACRONYMS

ADD	Average daily dose
ADD(life)	Average daily dose (lifetime)
ADD(year)	Average daily dose (year)
AhR	Aryl hydrocarbon receptor
ARAR	Applicable or relevant and appropriate requirement
AT	Averaging time
BW	Average body weight
CDC	Center for Disease Control
COPC	Chemical of potential concern
CSF	Cancer slope factor
CT	Central tendency exposure
ED	Exposure duration
EF	Exposure frequency
EP	Exposure period
EPC	Exposure point concentration
EPH	Extractable petroleum hydrocarbon
FDA	Food and Drug Administration
GC/MS	Gas chromatography/mass spectrometry
HBRA	Human Health Baseline Risk Assessment
HEAST	Health Effects Assessment Summary Tables
HI	Hazard index
HQ	Hazard quotient
IEUBK	Integrated Exposure Uptake Biokinetic
IR	Daily soil ingestion rate
IRIS	Integrated Risk Information System
LMS	Linearized multistage model
LOAEL	Lowest Observed Adverse Effect Level
MADEP	Massachusetts Department of Environmental Protection
MF	Modifying factor
MTBE	Methyl *tertiary*-butyl ether
NA	Not applicable (as specified in tables)
NCEA	National Center for Environmental Assessment
ND	Not detected (as specified in tables)
NH AGQS	New Hampshire Ambient Groundwater Quality Standards
NHDES	New Hampshire Department of Environmental Services
NHDHHS	New Hampshire Department of Health and Human Services
NOAEL	No observed adverse effect level
PAH	Polycyclic aromatic hydrocarbon
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo-p-dioxin
PDCF	Polychlorinated dibenzofuran
PEF	Particulate emission factor
PHC	Petroleum hydrocarbon
PRG	Preliminary remediation goal
PTWI	Provisional tolerable weekly intake
RAF	Relative absorption factor
RAGS	Risk Assessment Guidance for Superfund
RBC	Risk-based concentration

RCMP	Contaminated Sites Risk Characterization and Management Policy
RfC	Reference concentration
RfD	Reference dose
RI	Remedial investigation
RME	Reasonable maximum exposure
RPF	Relative potency factor
SHA	Sanborn, Head & Associates, Inc.
SIM	Selected ion monitoring
SSG	Soil Screening Guidance
SVOC	Semivolatile organic compound
SWRP	Surface water runoff pit
TDI	Tolerable daily intake
TEF	Toxicity equivalency factor
TEQ	Toxic equivalency quotient
TIC	Tentatively identified compound
TOC	Total organic carbon
TPH	Total petroleum hydrocarbons
UCL	Upper confidence limit
UF	Uncertainty factor
UR	Unit risk
USEPA	United States Environmental Protection Agency
USFWS	United States Fish and Wildlife Service
UST	Underground storage tank
VF	Volatilization factor
VPH	Volatile petroleum hydrocarbon
VOC	Volatile organic compound
WHO	World Health Organization

1.0 INTRODUCTION

This report presents the Human Health Baseline Risk Assessment (HBRA) for the Beede Waste Oil/Cash Energy Site (the "Site") in Plaistow, New Hampshire pursuant to the National Oil and Hazardous Pollution Contingency Plan (USEPA 1990). It complies with applicable New Hampshire Department of Environmental Services (NHDES) policy (NHDES, January 1998) and U.S. Environmental Protection Agency (USEPA) guidance (USEPA, 1989 and 1997a). This risk assessment evaluates cancer risk and non-cancer hazard associated with exposure to Site contamination for current and future uses of the Site. All risk estimates apply to site conditions in the absence of remediation or institutional controls that reduce or eliminate potential exposures.

The HBRA proceeds in four steps: Hazard Identification, Exposure Assessment, Toxicity Assessment, and Risk Characterization. The *Hazard Identification* briefly describes the extent of Site contamination and specifies the chemicals of potential concern (COPCs). The *Exposure Assessment* identifies potential exposure pathways for both current and future land use at the Site, estimates exposure point concentrations, and specifies exposure assumptions for estimating daily COPC intakes. The *Dose-Response Assessment* describes the available toxicity values for COPCs at the Site, any adjustments to toxicity values made in this assessment, and methods for evaluating toxicity for special case COPCs (i.e., PCBs, PAHs, lead and some metals). The *Risk Characterization* summarizes quantitative risk estimates by exposure scenario and describes exposure pathways that are evaluated qualitatively. The *Uncertainty Analysis* highlights uncertainties in the risk assessment that one should consider when interpreting and using results of this assessment.

1.1 Objective of the Human Health Baseline Risk Assessment

The HBRA quantifies, to the extent possible, current and future risk to human health resulting from contamination of the Site. This information can be useful in determining the need for and extent of any cleanup or other response action.

1.2 Site Background

The Site consists of two parcels (Parcel 1 and Parcel 2) that together comprise about 39 acres in a predominantly residential area of Plaistow, New Hampshire (Figure 1). Parcel 2 is the former location of a sand and gravel operation. It is now largely open space, but with several waste soil piles near its border with Parcel 1. Past operations primarily on Parcel 1 resulted in contamination of the Site. These activities included waste oil recycling, virgin oil distribution and storage, ethylene glycol recycling, and the storage of liquids and waste soils. Consequently, Site media (i.e. soil, groundwater, surface water, and sediment) are contaminated with petroleum hydrocarbons (PHCs), polychlorinated biphenyls (PCBs), chlorinated solvents, lead, and other metals. Some pesticides also have been detected at the Site, however, pesticide data for all Site media are questionable given analytical interferences, most likely due to PCB contamination. Contaminant concentrations in soil are heterogeneous across Parcel 1 given the presence of multiple source areas. Parcel 2 appears to be largely free of soil contamination, except near its border with Parcel 1.

There is one building on Parcel 1, close to the Site entrance, off Kelley Road at the northern boundary of the parcel. A smaller, older building was recently demolished. It was approximately 300 feet east of the main building and housed an ethylene glycol recycling operation. With the exception of the main building, most other structures, including approximately ninety above ground storage tanks, have been

removed from the Site. Most of Parcel 1 consists of exposed sandy soil with a narrow strip of forested area along the perimeter of the parcel. Two surface water runoff pits (SWRPs) are on Parcel 1. SWRP 1 is near the northern corner of the parcel, adjacent to an abutting residential property. SWRP 2 is south of the former building. A 140,000-gallon waste oil underground storage tank (UST) and several above ground storage tanks once occupied the area between the main building and the location of the former ethylene glycol recycling building. To the southwest of SWRP 2 is a large area of extensive soil staining where above ground waste oil tanks were once located. A former lagoon, filled in the early 1970s, is in the center of Parcel 1.

In previous years, the State of New Hampshire stocked Kelley Brook with trout downstream of the Site. Kelley Brook borders the Site after crossing under Kelley Road, flowing east along the northern edge of Parcel 1. At the northern edge of Parcel 1, free product has broken out in the wetland abutting the brook. An interceptor trench and recovery wells were installed in an attempt to control the breakout. Kelley Brook then continues along the western and northern edge of Parcel 2 and turns south. Near the northeastern edge of Parcel 2, an unnamed tributary to Kelley Brook crosses under Old County Road and joins Kelley Brook.

Residents surrounding the Site obtain their drinking water from private wells. Residential wells are sampled periodically by NHDES. Some of these private wells (wells on Lot 51-1-1 and Lot 32-3-4) have been impacted by volatile organic hydrocarbon (VOC) contamination, and treatment systems have been installed to remove these compounds from wells where New Hampshire Ambient Groundwater Quality Standards (AGQSs) have been exceeded. One residence adjacent to the Site (on Lot 32-3-11) is served by a bedrock well on Parcel 1 near the former building. To date, evidence of Site contamination has not been detected in this well.

This Site and adjacent properties have been the subject of several Agency for Toxic Substances and Disease Registry (ATSDR) public health consultations (ATSDR, 1996a, 1996b, 1998a; 1998b; 1998c).

1.3 Current and Future Land Use

Appendix A includes RAGS Part D Table 1 that summarizes how people might be exposed to Site contamination now and in the future. At present, the Site is not being used but is accessible to trespassers. Also, people might be exposed to Site contamination in Kelley Brook sediment, surface water, and fish. In the future, the Site may be developed for residential use.

2.0 HAZARD IDENTIFICATION

This section summarizes available data for quantifying potential risk at the Site and explains how COPCs were selected.

2.1 Data Sources

The HBRA relies on Site information and analytical data presented in the draft preliminary Remedial Investigation report (RI) prepared by Sanborn, Head & Associates, Inc. (SHA, 1999). We considered the following Site data:

- soil (surface soil and subsurface soil)
- Kelley Brook sediment
- Kelley Brook surface water
- groundwater (overburden, bedrock monitoring, and supply wells)
- Kelley Brook fish (brook trout, red fin pickerel, and crayfish)

The following sections provide a brief summary of data collected from the Site. Details of sampling design, sample collection efforts, and a general discussion of the data are available in the draft preliminary RI (SHA, 1999).

2.1.1 Soil

SHA collected surface and subsurface soil samples in several phases between October 1997 and May 1998. These samples were analyzed for metals, VOCs, semi-volatile organic compounds (SVOCs), volatile petroleum hydrocarbon/extractable petroleum hydrocarbon (VPH/EPH) fractions, PCBs (congeners, homolog groups and Aroclors), pesticides, and polycyclic aromatic hydrocarbons (PAHs). Four surface soil samples were analyzed for polychlorinated dibenzo-p-dioxin (PCDD) and polychlorinated dibenzofuran (PCDF) congeners in areas of high- and medium-level PCB contamination, where their concentrations might be highest.

Four of these soil samples were collected near Kelley Road (S-200, S-201, S-202 and S-203) and analyzed for PCBs to determine whether Site contamination could be impacting neighboring residences via fugitive dust emissions. In these samples, PCBs were used as a “marker” for Site contamination. In 1996, yard soil samples were collected from nine nearby residences and analyzed for PCBs. (ATSDR 1996b).

Three soil samples were collected from a portion of Parcel 1 where no waste oil handling or other activities were known to occur. These samples were analyzed to estimate Site-specific background conditions (S-141, S-142, and S-143) for naturally-occurring and anthropogenic contaminants.

2.1.2 Groundwater and Tap water

SHA collected groundwater samples between September 1997 and July 1998 using low-flow techniques from overburden and bedrock wells. Several samples were collected from monitoring wells, which contained free product oil. Site groundwater samples were analyzed most frequently for VOCs and metals. Some groundwater samples also were analyzed for PCB congeners, pesticides, VPH/EPH, SVOCs, and PAHs.

Tap water was collected from nearby residential wells potentially impacted by contaminated groundwater plumes originating at the Site. These samples were typically analyzed for VOCs. Also, all potentially impacted wells to the south of the Site were analyzed for a longer list of analytes, including PAHs, PCBs, and metals.

2.1.3 Sediment and Surface Water

SHA collected surface water and sediment samples (0-1 ft depth) from Kelley Brook in October 1997 from the stream channel and adjacent wetlands. Sediment sampling locations represent depositional areas. Eleven unfiltered surface water samples and twenty sediment samples were collected and analyzed for SVOCs, PAHs, VPH/EPH, PCBs, VOCs, pesticides, and metals. In May 1998, seven additional sediment samples were collected for two purposes:

- 4 samples to better define the spatial extent of contamination from the oil breakout area; and
- 3 samples to test a different VOC sampling method.

Additional sediment samples (0-0.25 ft) were collected in 1999 as part of an investigation regarding vegetation die back in Kelley Brook. These sediment samples were not used to estimate human exposure to Site-related contamination in the brook.

2.1.4 Fish and Crustaceans

Personnel from the U.S. Fish and Wildlife Service (USFWS) and NHDES collected fish from four reaches of the brook, KB-1, KB-2, KB-3, and KB-4, in August 1996 (Figure 2). Reaches KB-4 and KB-3 are upstream of the Beede Waste Oil Site. Reach KB-4 is upstream of the Rt. 121 A culvert. Reach KB-3 extends from the Rt. 121 A culvert to the Kelley Road culvert. Reach KB-2 runs through the Beede Waste Oil Site, from the Kelley Road culvert to the Rt. 125 culvert. Reach KB-1 is downstream of the Site, extending from the Rt. 125 culvert to the confluence of Kelley Brook with the Little River. Nearly all, if not all, fish in these reaches were collected using electroshocking (Ms. Susan Svirsky, USEPA Region 1, personal communication). No crayfish were collected from KB-2, the reach adjacent to the Site.

Whole fish samples of brook trout, red fin pickerel, and crayfish were analyzed for PCB congeners, dioxin/furan congeners, pesticides, and metals. Figures 3, 4, and 5 show total metal, pesticide, and PCB concentrations in each species by reach. These plots do not include dioxin congeners because only brook trout and red fin pickerel from KB-2 and crayfish from KB-1 were analyzed for these congeners, with only 2,3,7,8-TCDF detected in brook trout and red fin pickerel.

2.2 Data Quality

O'Reilly, Talbot & Okun performed Tier II or Tier III (for dioxins and PCB congeners) data validation on all samples used to estimate risk except for the fish tissue data. Screening-level data guided selection of analytes of interest for validated samples used in the risk assessment. For detailed evaluation of data quality, see the series of data validation reports prepared by O'Reilly, Talbot & Okun. We adhered to all recommendations in these reports regarding data useability for the purpose of screening COPCs and calculating exposure point concentrations (EPCs).

Site data are generally of high quality, although the desired detection limits were not attained in all media for all analytes. High detection limits occurred for many COPCs in soil (VOCs, SVOCs, pesticides) in

areas with high levels of PCB or petroleum contamination. Pesticide data are particularly problematic, likely due to analytical interference of PCBs. As a result, many pesticides are groundwater COPCs, but these detected concentrations may be false positives. Appendix B includes "Data Useability Worksheets" that discuss data quality issues for each medium in greater detail.

2.3 Data Management

Duplicate sample results were averaged. If a chemical was not detected in either sample, the lower of the two detection limits was used. If a chemical was not detected in one sample and detected in the other, the detected concentration was used. Results from multiple samples collected at the same location were averaged, following the same protocol described for duplicates.

2.3.1 Soil and Sediment Data

Total PCBs

The concentration of total PCBs in soil was calculated by summing the PCB chlorination level concentrations (e.g., mono-, di, and trichlorobiphenyls). We subtracted dioxin-like PCB congener concentrations and assumed that non-detected compounds were present at 1/2 the detection limit. Where PCB chlorination level data were not available, we used the sum of the detected concentrations of PCB Aroclor data (quantified primarily as Aroclor 1242, with some Aroclor 1260) as the total PCB concentration.

Results from Multiple Analytical Methods

Naphthalene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, and 1,2-dichlorobenzene were analyzed by more than one analytical method. The exposure point concentration of these chemicals was calculated by averaging the results from all methods using the following methodology:

1. For naphthalene, the validator compared the results of selected ion monitoring (SIM) and USEPA Method 8270 analyses and recommended which data to use. The recommended data were then averaged with USEPA Method 8260 results. If naphthalene was detected using one method, but not the others, the detected concentration was chosen. If naphthalene was not detected using any of the three analytical methods, ½ of the lowest detection limit was used to estimate the chemical concentration.
2. For dichlorobenzenes, Method 8270 and 8260 results were averaged if detected by both methods. If the chemical was detected using one method, but not the other, the detected concentration was chosen. If the chemical was not detected using either method, ½ of the lowest detection limit was used to estimate the chemical concentration.

2.3.2 Groundwater Data

Ethylene dibromide and 1,2-dibromo-3-chloropropane were analyzed by two methods: 8260A and 504.1. Method 504.1 was used to achieve detection limits lower than drinking water criteria for these two chemicals. Where 504.1 data were available, they were used. The following wells were resampled due to a question of laboratory contamination with methylene chloride: AE-10RS, AE-21RS, AE-22RS, SH-32RS, SH-33RS, and SH-57RS. We averaged data from these two sampling rounds because the validator did not find any analytical reason to exclude the data from the first sampling round.

2.4 Chemicals of Potential Concern (COPCs)

COPCs are chemicals retained for further evaluation in the risk assessment. RAGS Part D Table 1 (Appendix A) lists the potential human exposure pathways used to select COPCs at the Site. Appendix B includes Toxicological Profiles for COPCs.

Table 1 summarizes the COPCs for each medium. RAGS Part D Tables 2.1 through 2.14 (Appendix D) provide the Site concentrations and screening criteria (e.g., risk-based concentrations (RBCs), applicable or relevant and appropriate (ARAR) criteria) used to select COPCs. The following subsections explain how COPC screening was performed for the six environmental media sampled at the Site (tap water, groundwater, surface water, soil, sediment, and fish). Every effort was made to achieve detection limits below applicable screening criteria and ARARs. However, some VOCs, SVOCs, and pesticides were infrequently or never detected, but have maximum detection limits greater than screening criteria. Elevated detection limits occur in soil and groundwater samples collected from Parcel 1 near source areas where concentrations of all chemicals tend to be highest. We consider the impact of this problem on COPC screening.

2.4.1 Groundwater and Tap Water

Groundwater and tap water data were screened against USEPA Region III Risk-Based Concentrations for tap water ingestion (1999) and against the New Hampshire Ambient Groundwater Quality Standards (NH AGQS) (See Tables 2.4, 2.8, 2.9, and 2.10). Some chemicals in VPH/EPH fractions were quantified individually as well as part of the fractions. Therefore, we screened these chemicals individually, unless otherwise noted. Chemicals with detection frequencies less than 5% and with maximum concentrations less than RBCs and ARARs are excluded from the COPC list. Some chemicals with less than 5% detection frequencies were measured above RBCs, but they are excluded from the COPC list because they are present in so few samples. Chemicals with maximum concentrations above the applicable ARAR are retained on the COPC list, regardless of detection frequency. Some chemicals were analyzed in groundwater with detection limits that frequently exceeded RBCs and ARARs (Table 2). However, these chemicals were never detected despite relatively low detection limits, even in the more contaminated areas of Parcel 1. Therefore, they do not appear to be important contaminants at the Site and were not retained as COPCs.

Chloride and iron were not selected as COPCs; however, these chemicals may cause aesthetic concerns if Site groundwater is used for drinking water in the future. USEPA National Secondary Drinking Water Standards are non-enforceable guidelines regulating contaminants that may cause cosmetic effects (e.g., skin or tooth discoloration) or aesthetic effects (i.e. taste, odor, or color) (USEPA Office of Water website, 3/99). The maximum chloride concentration (390 mg/L) exceeds the USEPA Secondary Drinking Water Standard of 250 mg/L. The maximum iron concentration (110 mg/L) greatly exceeds the EPA secondary standard for iron (0.3 mg/L).

Table 1. Summary Of Chemicals Of Potential Concern (COPC) By Medium/Exposure Pathway (1)										
CAS Number	**Chemical**	**Soil** **Ingest** **Inhale (part/vapor)** **Dermal** Current and future resident	 future worker	**Ground water** **Ingest** **Inhale** **Dermal** future worker	**Ground water** **Ingest** **Inhale** **Dermal** future resident	**Ground water** **Ingest** **Inhale** **Dermal** current resident (2)	**Ground water** **Vapor intrusion (Inhale)** future resident	**Sediment** **Ingest** **Dermal** future resident	**Surface Water** **Ingest** **Dermal** future resident	**Fish** **Ingest** future resident
	Inorganics									
7440-36-0	Antimony	X	X	X	X					
7440-38-2	Arsenic	X	X	X	X			X	X	X
7440-39-3	Barium	X	X	X	X					
7440-41-7	Beryllium	X	X					X		
7440-43-9	Cadmium	X	X	X	X					
7440-47-3	Chromium	X	X	X	X			X		X
7440-50-8	Copper	X								X
7439-92-1	Lead	X	X							X
7439-96-5	Manganese	X		X	X			X	X	X
7439-97-6	Mercury	X	X					X		X
7440-02-0	Nickel	X	X							
7782-49-2	Selenium			X	X					
7440-28-0	Thallium							X		
7440-62-2	Vanadium	X								
7440-66-6	Zinc	X	X							X
7439-98-7	Molybdenum	X							X	
	Nitrate-N			X	X					
	VOCs									
75-71-8	Dichlorodifluoromethane									
74-87-3	Chloromethane			X	X					
75-01-4	Vinyl chloride			X	X	X	X		X	
75-00-3	Chloroethane			X	X		X			
75-35-4	1,1-Dichloroethene			X	X		X			
75-09-2	Methylene chloride			X	X					
1634-04-4	Methyl t-butyl ether			X	X			X		
156-60-5	trans-1,2-Dichloroethene			X	X		X			
75-34-3	1,1-Dichloroethane			X	X					
156-59-2	cis-1,2-Dichloroethene	X	X	X	X	X	X		X	
71-55-6	1,1,1-Trichloroethane			X	X					
107-06-2	1,2-Dichloroethane			X	X	X				
71-43-2	Benzene			X	X	X				
79-01-6	Trichloroethene			X	X	X	X			
108-88-3	Toluene			X	X					
127-18-4	Tetrachloroethene	X	X	X	X					
100-41-4	Ethylbenzene			X	X					
79-34-5	1,1,2,2-Tetrachloroethane			X	X					
103-65-1	n-Propylbenzene	X	X	X	X					
108-67-8	1,3,5-Trimethylbenzene	X	X	X	X					
98-06-6	tert-Butylbenzene	X	X	X	X					
95-63-6	1,2,4-Trimethylbenzene	X	X	X	X					
135-98-8	sec-Butylbenzene	X	X	X	X					
25155-15-1	p-Isopropyltoluene (cymene)	X	X	X	X					
104-51-8	n-Butylbenzene	X	X	X	X					
95-50-1	1,2-Dichlorobenzene			X	X					

Table 1. Summary Of Chemicals Of Potential Concern (COPC) By Medium/Exposure Pathway (1)										
CAS Number	**Chemical**	**Soil** **Ingest Inhale (part/vapor) Dermal** Current and future resident	 future worker	**Ground water Ingest Inhale Dermal** future worker	**Ground water Ingest Inhale Dermal** future resident	**Ground water Ingest Inhale Dermal** current resident (2)	**Ground water Vapor intrusion (Inhale)** future resident	**Sediment** **Ingest Dermal** future resident	**Surface Water Ingest Dermal** future resident	**Fish** **Ingest** future resident
91-20-3	Naphthalene	X	X	X	X					
	PAHs									
91-57-6	2-Methylnaphthalene			X	X					
208-96-8	Acenaphthylene								X	
85-01-8	Phenanthrene								X	
56-55-3	Benz(a)anthracene	X	X					X	X	
218-01-9	Chrysene	X	X					X	X	
205-99-2	Benzo(b)fluoranthene	X	X					X	X	
207-08-9	Benzo(k)fluoranthene	X	X					X	X	
50-32-8	Benzo(a)pyrene	X	X					X	X	
193-39-5	Indeno(1,2,3-cd)pyrene	X	X					X	X	
53-70-3	Dibenz(a,h,)anthracene	X	X					X	X	
191-24-2	Benzo(g,h,i)perylene								X	
	SVOCs									
117-81-7	bis(2-Ethylhexyl)phthalate	X	X							
	Volatile Petroleum Hydrocarbons									
	C5-C8 Aliphatics						X			
	C9-C12 Aliphatics						X			
	C9-C10 Aromatics			X	X					
	Extractable Petroleum Hydrocarbons									
	C9-C18 Aliphatics	X	X							
	C11-C22 Aromatics	X	X	X	X			X		
	Total PCBs	X	X					X		X
	Dioxin TEQ	X	X					X		X
	Pesticides									
309-00-2	Aldrin			X	X					X
319-84-6	Alpha-BHC (hexachlorocylcohexane (HCH))			X	X					
58-89-9	Gamma-BHC (hexachlorocylcohexane (HCH), lindane)			X	X					
76-44-8	Heptachlor			X	X	X				
1024-57-3	Heptachlor epoxide			X	X				X	X
72-55-9	4,4'-DDE									X
50-29-3	4,4'-DDT									X
60-57-1	Dieldrin			X	X					X
5103-73-1	Cis-nonachlor									X
39765-80-5	Trans-nonachlor									X

Notes:
(1)This table summarizes COPCs screened in RAGS Part D Tables 2.1 through 2.14 (Appendix D).
(2) This table summarizes COPCs screened in RAGS Part D Table 2.4, however it does not include dichlorodifluoromethane. Dichlorodifluoromethane was detected once in a well on lot 32-2-5. However, only untreated groundwater samples from wells on lots 51-1-1 and 32-3-4 were used to estimate groundwater exposure for the current resident.

Table 2. Chemicals That Were Not Detected in Groundwater and Were Therefore Not Retained as COPCs, but Have Detection Limits Greater than USEPA Region III Risk-Based Concentrations (RBCs) and/or NH Ambient Groundwater Quality Standards (AGQS)

Chemical	detection limit range (µg/L)	detection frequency	USEPA Region III RBC for Tap water (RBC) (µg/L)	NH AGQS (ARAR) (µg/L)	frequency with which detection limits exceed RBC	frequency with which detection limits exceed ARAR
METALS						
Molybdenum	100	0/10	18	-	10/10	-
Thallium	2	0/97	0.29	2	97/97	0/97
Vanadium	50	0/10	26	-	10/10	-
VOCs						
Bromomethane	2 - 20	0/129	0.85	10	129/129	4/129
Chloroform	2 - 20	0/129	0.15	6	129/129	8/129
Carbon tetrachloride	2 - 20	0/129	0.16	5	129/129	8/129
1,2-Dichloropropane	2 - 20	0/129	0.16	5	129/129	8/129
Carbon Disulfide	2 - 20	0/129	100	7	0/129	5/129
Tetrahydrofuran	10 - 100	0/121	8.8	150	121/121	0/121
Dichlorobromomethane	2 - 20	0/129	0.17	0.3	129/129	129/129
cis-1,3-Dichloropropene	2 - 20	0/129	0.077	0.2	129/129	129/129
Trans-1,3-Dichloropropene	2 - 20	0/129	0.077	0.2	129/129	129/129
1,1,2-Trichloroethane	2 - 20	0/129	0.19	5	129/129	8/129
Dibromochloromethane	2 - 20	0/129	0.13	0.3	129/129	129/129
Bromoform	2 - 20	0/129	8.5	4	5/129	8/129
1,1,1,2-Tetrachloroethane	2 – 20	0/129	0.41	70	129/129	0/129
1,2,3-Trichloropropane	2 - 20	0/129	0.0015	40	129/129	0/129
2-Chlorotoluene	2 - 20	0/128	12	100	3/128	0/128
1,2-Dibromo-3-chloropropane	0.02 - 20	0/129	0.047	0.2	49/129	49/129
Hexachlorobutadiene	2 - 20	0/129	0.86	0.5	129/129	129/129
Ethylene dibromide	0.02 - 20	0/129	0.00075	0.05	129/129	49/129
PAHs						
Benz(a)anthracene	0.01-0.054	0/10	0.092	0.05	0/10	3/10 (0.054 vs. 0.05)
Benzo(b)fluoranthene	0.01-0.054	0/10	0.092	0.05	0/10	3/10 (0.054 vs. 0.05)
Benzo(a)pyrene	0.01-0.054	0/10	0.0092	0.2	10/10	0/10
Indeno(1,2,3-cd)pyrene	0.01-0.054	0/10	0.092	0.05	0/10	3/10 (0.054 vs. 0.05)
Dibenz(a,h)anthracene	0.01-0.054	0/10	0.0092	0.005	10/10	10/10
SVOCs						
bis(2-Chloroethyl)ether	2	0/10	0.0096	10	10/10	0/10
1,3-dichlorobenzene	2	0/10	0.55	600	10/10	0/10
bis(2-Chloroisopropyl)ether	2	0/10	0.26	300	10/10	0/10
Hexachloroethane	2	0/10	4.8	1.9	0/10	10/10
N-Nitroso-di-n-propylamine	2	0/10	0.0096	-	10/10	-
Nitrobenzene	2	0/10	0.35	-	10/10	-
4,6-Dinitro-2-methylphenol	5	0/10	0.37	-	10/10	-
Hexachlorobenzene	1	0/10	0.042	1	10/10	0/10
Pentachlorophenol	1	0/10	0.56	1	10/10	0/10
3,3'-Dichlorobenzidine	5	0/10	0.15	1.3	10/10	10/10
PESTICIDES						
Toxaphene	0.099-0.1	0/10	0.061	3	10/10	0/10

2.4.2 Soil

We screened maximum chemical concentrations in soil against appropriate screening criteria (Region III RBCs for residential soil for the residential exposure scenario (1999), RBCs for industrial soil for the future construction worker scenario (1999), and NHDES S-1 soil standards) (See Tables 2.5, 2.6, 2.7, 2.11, 2.12, 2.13, and 2.14). Some chemicals in VPH/EPH fractions were quantified individually as well as part of the fractions. Therefore, we screened these chemicals individually, unless otherwise noted. Chemicals detected less than 5% of the time or with maximum concentrations less than RBCs and ARARs are excluded from the COPC list. If the maximum concentration of a chemical exceeds the ARAR or RBC and it is detected greater than 5% of the time, it is included as a COPC. Appendix E includes bar plots showing the distribution of soil COPC concentrations for all sampling locations where the COPC was detected. On these plots, contaminant concentrations are compared to relevant screening criteria and Site-specific background. Maximum COPC concentrations frequently exceed Site-specific background concentrations.

The COPC screening for soil is based on the entire validated data set (i.e. samples at all depths). However, no compounds screened in based on concentrations in soil samples greater than ten feet deep. If we had screened COPCs using just the surface soil data (0-1 ft depth), several compounds would have screened out: copper, vanadium, molybdenum, cis-1,2-dichloroethene (cis-1,2-DCE), naphthalene, and the alkylbenzenes (n-butylbenzene, n-propylbenzene, 1,3,5-trimethylbenzene, tert-butylbenzene, 1,2,4-trimethylbenzene, sec-butylbenzene, and p-isopropyltoluene).

Cyanide was detected at 69 mg/kg in one soil sample near the former landfill area in sample TP127/139. This concentration does not exceed the USEPA Region III RBC for free cyanide of 1,600 mg/kg or the NHDES S-1 standard for free cyanide of 100 mg/kg. However, it does exceed RBCs for some other forms of cyanide. Whether cyanide is included as a COPC depends on its chemical form and the extent of cyanide contamination at the Site. SHA measured reactive cyanide and sulfide in surface soils with visible contamination. These data were not validated, but they do not indicate the presence of cyanide. Also, NHDES sampled 9 wells near the former landfill area during the summer of 1999. Cyanide was not present in groundwater from these wells. For all of these reasons, cyanide is not likely to be a widespread contaminant at the Site and is not retained as a COPC.

Screening-level soil gas measurements were collected across the Site in 1995, and detected concentrations consisted primarily of 1,1,1-trichloroethane (TCA), with lesser concentrations of TCE, PCE and occasional detected concentrations of 1,1-DCA, 1,1-DCE, and cis-1,2-DCE. (SHA, 1995, Figure 7). More recent screening-level soil gas measurements under the new Site building found a similar pattern of contamination with a maximum TCA concentration of 940 ppb, and maximum concentrations of chlorobenzene and PCE of 260 and 80 ppb, respectively. Based on groundwater data, TCA was not retained as a COPC for the “soil gas vapor intrusion pathway” into future residences. Soil gas data were not used to quantify risk to a future resident (See discussion in Section 3.1.1), but they are considered qualitatively in the risk characterization for this pathway.

Carcinogenic PAHs were screened as a group. If benzo(a)pyrene (B(a)P) screened in, we screened in the rest of the carcinogenic PAHs given their similar mechanism of toxicity (USEPA, 1993).

2.4.3 Sediment

We screened maximum chemical concentrations in sediment against appropriate toxicity screening criteria (Region III RBCs for residential or industrial soil, 1999) and ARARs (NHDES S-1 standards) (See Table 2.1). Some chemicals in VPH/EPH fractions were quantified individually as well as part of the fractions. Therefore, we screened these chemicals individually, unless otherwise noted. Chemicals with maximum concentrations less than RBCs are excluded from the COPC list. Some chemicals with less than 5% detection frequencies were measured above screening criteria, but they are excluded because they are present in so few samples.

Carcinogenic PAHs were screened as a group. If benzo(a)pyrene (B(a)P) screened in, we screened in the rest of the carcinogenic PAHs given their similar mechanism of toxicity (USEPA, 1993).

2.4.4 Surface water

We screened maximum chemical concentrations in surface water against appropriate toxicity screening criteria (Region III RBCs for tap water, 1999) and ARARs (NH Water Quality Criteria) (See Table 2.2). Some chemicals in VPH/EPH fractions were quantified individually as well as part of the fractions. Therefore, we screened these chemicals individually, unless otherwise noted. Chemicals with maximum concentrations less than RBCs and ARARs were excluded from the COPC list. Some chemicals with less than 5% detection frequencies were measured above the RBC, but were excluded because they are present in so few samples. However, if the maximum concentration of a chemical exceeds the ARAR, it is included as a COPC regardless of detection frequency.

2.4.5 Fish

We screened maximum chemical concentrations in trout and pickerel tissue against appropriate toxicity screening criteria (Region III RBCs for fish ingestion, 1999) and Food and Drug Administration (FDA) Action Limits (See Table 2.3). Chemicals detected less than 5% of the time or with maximum concentrations less than toxicity screening criteria are excluded from the COPC list. Some chemicals with less than 5% detection frequencies were measured above the toxicity screening criteria, but they are still excluded because they are present in so few samples.

2.4.6 Dioxin TEQ

Four soil samples were analyzed for dioxin congeners. Samples analyzed for dioxin congeners were collected from locations with mid- to high-level PCB concentrations to represent the probable upper end of the range of dioxin concentrations. Dioxin-like PCB congeners and dioxin congeners were screened collectively as a dioxin Toxic Equivalency Quotient (TEQ). We calculated TEQs for dioxins and dioxin-like chemicals in each medium by multiplying the detected concentration (or half the detection limit) of each dioxin or dioxin-like PCB congener by its Toxic Equivalency Factor (TEF) and adding these TEFs to obtain the dioxin TEQ (WHO, 1998). The TEQ calculated from maximum soil concentrations of dioxin congeners alone is 0.095 ppb, approximately 10 times lower than the USEPA recommended cleanup level of 1 ppb in soil for residential property (USEPA 1998a). However this TEQ is approximately 200 times higher than the Region III RBC; therefore, dioxin TEQ is retained as a COPC.

2.4.7 Tentatively Identified Compounds

Over one hundred tentatively identified compounds (TICs) are reported in soil. Most of the TICs are petroleum hydrocarbons, which are quantified as part of the VPH/EPH fractions. Therefore, risk associated with these TICs is quantified as part of the risk from the VPH/EPH fractions. It is difficult to quantify risk from the non-petroleum hydrocarbon TICs due to a lack of toxicity values for these compounds. However, TICs are reported for samples that already contain high concentrations of COPCs.

2.4.8 Comparison of the Validated Data Set and the Screening Level Data Set for Soil

With the exception of fish data, only validated data were used in this assessment. The validated data set includes samples collected from the principal identified source areas. As a check on the consistency of the validated data set with the larger screening level data set, we compared the maximum COPC concentrations in the following data sets:

- Screening-level Phase I (0-1 ft depth) soil data from Parcel 1
- Validated (0-1 ft depth) soil data used in the risk assessment (all Parcel 1 samples and three Parcel 2 samples).

The maximum detected concentrations in the validated data set exceed the maximum detected concentrations in the screening-level data set except for arsenic, cis-1,2-DCE, and tetrachloroethene. Thus, some high concentrations of these compounds were “missed” by using only the validated data in the risk assessment. However, these three compounds were retained as COPCs using the validated data set.

Many of the highest lead concentrations are in the screening level data set. However, these high concentrations are reflected in the validated data set, specifically in Sample S-223 that has very high concentrations of lead as well as other COPCs. Sample S-223 is an outlier among validated data, but it is not an outlier among all screening and validated data.

3.0 EXPOSURE ASSESSMENT

The exposure assessment estimates the magnitude of actual and potential human exposures, the frequency and duration of these exposures, and the pathways by which people are potentially exposed for both current and future land use at the Site. Exposure estimates are based on both measured and modeled concentrations. This section describes potentially complete exposure pathways at the Site, the approach used to calculate EPCs, and the exposure assumptions and models used to calculate daily COPC intake estimates.

The reasonable maximum exposure (RME) is the highest exposure that is expected to occur at the Site and is representative of a high-end risk. The RME approach uses high-end values from exposure parameter distributions to arrive at an upper-bound risk estimate. The central tendency (CT) approach uses average values for exposure parameters and, thus, yields estimates of average risk to an individual.

One acre is the minimum lot size for new residential developments in Plaistow, New Hampshire. We consider this one acre lot size in deciding whether "hot spots" of contamination exist at the Beede Waste Oil/Cash Energy Site (see Section 3.3.1).

3.1 Potential Exposure Pathways

Site-related contamination is present in Site soils and groundwater as well as Kelley Brook sediment, surface water, and biota and nearby private residential drinking water wells. Also, COPCs might be present in fugitive dust emanating from the Site. In the future, vegetation in home gardens planted by on-Site residents could take up COPCs.

RAGS Part D Table 1 (Appendix A) summarizes potential exposure pathways. An exposure pathway is complete if there is a source or contaminant release from a source, an exposure point where contact can occur, and an exposure route by which contact can occur. Exposure pathways are identified for potentially exposed populations by considering the source of contaminants, locations of contaminants or exposure points, and the likelihood of exposure to the contaminants at the exposure points.

We evaluate six exposure scenarios: a future resident, a current resident living near the Site, a child playing in Kelley Brook, a trespasser, an adult fishing in Kelley Brook, and an outdoor construction worker. Exposure assumptions for each exposure scenario are presented in RAGS Part D Table 4s (Appendix J).

3.1.1 Future resident

We assume that the future land use of the Site could be residential and evaluate the potential exposure of a future resident to contamination in soil and groundwater. We evaluate exposure to an adult and a child resident separately. Future residents could be exposed to Site contamination via the following pathways:

- Dermal contact with, ingestion and inhalation of groundwater;
- Dermal contact with, ingestion and inhalation of soil; and
- Ingestion of home garden produce that takes up soil contaminants.

Dermal contact with and ingestion of groundwater

Future on-Site child and adult residents might be exposed to Site contaminants by drinking, bathing, and otherwise using contaminated groundwater. We quantify risk associated with exposure to COPC concentrations in future on-Site wells via ingestion and dermal contact (Tables 4.12 and 4.13).

Inhalation from groundwater

Residents can also be exposed to contaminants that volatilize from tap water during showering and during other household uses (e.g., Wilkes and Small, 1992). We qualitatively assess inhalation exposure to volatile COPCs in tap water in accordance with USEPA Region I policy (1995). Specifically, USEPA Region I recommends assuming that risk from this exposure pathway is equal to risk from the ingestion pathway for each volatile COPC.

Vapor intrusion of groundwater contaminants into residences

Soil gas samples collected in 1997 contain several contaminants: 1,1,1-TCA (50 to 940 ppb), tetrachloroethene (60-80 ppb), and chlorobenzene (260 ppb) (SHA, 1995). None of these data are validated and were not used to estimate risk from a vapor intrusion pathway into future residences.

Any risk associated with this exposure pathway is likely to contribute negligibly to risk associated with exposure to contaminated groundwater brought into the residence for use as tap water. Also, there is a great deal of uncertainty associated with predicting risk from vapor intrusion for buildings that have not been built. For these reasons, risk associated with this pathway was not quantified for a future resident. Before any residences are built on the Site, groundwater and soil contamination must be remediated so that groundwater meets all drinking water standards. This effort is likely to reduce the potential for exposure by the vapor intrusion pathway. However, if soil remediation is completed and a clean groundwater source is provided for drinking water, the risk to a future resident due to vapor intrusion from groundwater will have to be quantified.

Dermal contact with and ingestion of soil

Adult residents may ingest soil or contact it dermally while using their yards for recreation and gardening. Children may also be exposed to soil via ingestion and dermal routes of exposure while walking, biking, or otherwise playing. Their exposure is likely to be more intensive than adult resident exposure.

We evaluate exposure of the future resident to two separate vertical soil strata: zero to ten feet and zero to one foot (i.e. surface soil). Following USEPA guidance, we assume vertical mixing of soil in the future and evaluate exposure from the surface down to a depth of 10 feet, which EPA considers the vertical limit of excavation for building a foundation. While there are areas of subsurface contamination at the Site, much of the contamination is at the surface. Therefore, we also evaluate potential exposure to 0-1 ft soil only.

We estimate exposure for the child and the adult resident scenarios quantitatively, estimating exposure to 0 to 1 foot soils and 0 to 10 feet soils separately (Tables 4.15, 4.16, 4.17, and 4.18).

Inhalation of fugitive dust and vapors from soil

For the adult resident, we assume that inhalation of fugitive dust and volatilized contaminants from surface soil or subsurface soil, exposed as a result of excavation work, occurs while walking and gardening. Exposure via inhalation of fugitive dust and vapors occurs for the child resident while walking, biking, or playing. We evaluate both the child and the adult resident scenarios quantitatively using modeled EPCs (see section 3.3.6) (Tables 4.20, 4.21, 4.22, 4.23, 4.25, 4.26, 4.27, 4.28). Chemical-specific risk estimates are not presented for these pathways, however, the contribution of these pathways

to total Site risk are shown in RAGS Part D Tables 7, 8, 9 and 10 and Section 5.3 of this report. They contribute a small fraction of estimated risk for the future resident.

Ingestion of homegrown garden produce

A future adult and child resident may be exposed to COPCs in soil while gardening and by consuming produce grown in the garden that has taken up COPCs from soil. Some contaminants, particularly metals, may be taken up into the plant tissue. There are few data available for plant uptake of organic compounds. For many organic compounds, including PCBs, concentrations measured in vegetation are largely attributed to the aerial deposition rather than translocation (e.g., Smith and Jones, 2000). Modeling uptake for each COPC at the Site would be a complex and uncertain task, and we have not attempted it in this report. The importance of this pathway depends on ingestion rates for home garden produce and whether residents wash off potentially contaminated soil prior to eating produce.

3.1.2 Current resident

Current child and adult residents living near the Site might be exposed to COPCs by drinking, bathing, and otherwise using contaminated groundwater. Residents living near the Site may potentially be exposed to fugitive dust from the Site.

Dermal contact with and ingestion of groundwater

We quantify risk associated with exposure to COPC concentrations in existing wells via ingestion and dermal contact (Tables 4.6 and 4.7).

Inhalation from groundwater

Neighboring residents use groundwater for all typical household uses (drinking, bathing, and washing dishes). We qualitatively assess inhalation exposure to tap water COPCs in accordance with USEPA Region I policy (1995). We assume that the exposure and risk from the inhalation pathway is equal to that of the ingestion pathway for VOCs in tap water.

We do not quantify risk associated with potential vapor intrusion of groundwater contaminants into existing residences given the depth to groundwater contamination near these homes. No Site contaminants were detected in historic screening-level soil gas measurements collected near current residents.

In August 1997, NHDES collected headspace air samples above a shallow dug well in the basement of a residence adjacent to Parcel 1 near SWRP 1. While residents no longer drink from the shallow well, their home might be impacted from volatile contaminants impacting indoor air quality. In response to this concern, NHDES and NHDHHS collected an air sample from the well headspace. NHDHHS concluded that detected concentrations are “below levels of concern for human health risk from inhalation.” (ATSDR, 1998a). NHDHHS further noted that sampling over time in this well indicates a downward trend in concentrations, and all detected contaminants are below drinking water standards.

Exposure of neighboring residents to fugitive dust

This pathway is evaluated qualitatively using data collected from nine nearby residential backyards and four soil samples (S-200, S-201, S-202, and S-203) collected near Kelley Road to determine whether Site contamination could be impacting neighboring residences.

3.1.3 Children playing in Kelley Brook (Recreational use of Kelley Brook)

Children could play in Kelley Brook now or in the future. For this scenario, we assume that 6 to 18 year olds wade, fish, or play in Kelley Brook two to five times per week from May to September (Tables 4.1 and 4.3).

Dermal contact with and ingestion of surface water

Dermal exposure to surface water occurs when the child wader might also accidentally ingest a small amount of surface water. We assume the child wader ingests 50 mL, or about one mouthful of water.

Dermal contact with and ingestion of sediment

The child wader might also be exposed to sediment via dermal and ingestion routes of exposure when standing in shallow water or playing on the bank.

3.1.4 Current trespasser

There is evidence of trespassers using the Site, including graffiti and a sign warning against dirt biking in some areas. For this scenario, we assume that 6 to 18 year old adolescents walk, bike, or play two to five times per week from May to September. Trespassers may be exposed to soil through ingestion, dermal contact, and inhalation of fugitive dust and volatilized contaminants (Tables 4.8, 4.9, 4.10). We evaluate all of these pathways quantitatively, however, we estimate exposure from fugitive dust and volatilized contaminants using modeled instead of measured EPCs (see section 3.3.6). Chemical-specific risk estimates are not presented for the fugitive dust and vapor inhalation pathways, however the contribution of these pathways to total Site risk are shown in RAGS Part D Tables 7, 8, 9 and 10 (See Appendices N, O, P, and Q, respectively) and Section 5.3 of this report.

3.1.5 Adult fisherperson at Kelley Brook

For this scenario, we assume that an adult fishes one to three times per week for five non-winter months (May – September). The fisherperson might be exposed to Site contamination by contacting and incidentally ingesting sediment and surface water during fishing and by consuming fish from Kelley Brook (Tables 4.2, 4.4, 4.5).

Dermal contact with and ingestion of surface water

Dermal exposure to and incidental ingestion of surface water occurs when wading in Kelley Brook while fishing.

Dermal contact with and ingestion of sediment

A fisherperson may also be exposed to sediment via ingestion and dermal contact when wading in Kelley Brook while fishing.

Fish consumption

We assume that the fisherperson only eats the brook trout that they catch in Kelley Brook. Chemrisk (1991) administered a questionnaire to recreational anglers in Maine to determine their freshwater fish consumption habits. Anglers reported a mean consumption rate of 3.7 grams/day and an upper 95th percentile consumption rate of 12 g/d for fish caught in rivers and streams. We assume the fisherperson consumes fish at the 12 g/d rate and that all of the fish are caught in Kelley Brook.

3.1.6 Future outdoor construction worker

Outdoor construction workers are likely to be exposed to Site contamination during future excavation work via dermal, ingestion, and inhalation (fugitive dust and vapor) exposure to soil and possibly groundwater (Tables 4.11, 4.14, 4.19, 4.24). The construction worker is exposed for 3 to 5 months per year.

Dermal contact with and ingestion of groundwater

We assume that a construction worker will not be exposed to groundwater deeper than 15 feet below ground surface. This cutoff was selected assuming workers might dig to ten feet and that groundwater elevations might fluctuate by as much as ±5 feet. We assume that the construction worker could incidentally ingests 50 mg/L of groundwater when working at an excavation site.

Inhalation from Groundwater

Risk associated with inhalation of volatile COPCs in groundwater was not quantified. Many assumptions are required to estimate COPC concentrations in a construction trench, and risk associated with dermal exposure to groundwater is very high for the construction worker. Therefore, estimating this additional and uncertain estimate of risk would provide little information.

Dermal contact with and ingestion of soil

For the outdoor worker, we assume that dermal exposure with and incidental ingestion of contaminants in surface soil or subsurface soil occurs during excavation work. The construction worker may be exposed to contaminants in surface soil and in soil to the depth of excavation. We assume the depth of excavation to be 10 feet.

Inhalation of fugitive dust and vapors from soil

For the outdoor worker, we assume that inhalation of fugitive dust and vapors from surface soil or subsurface soil occurs during excavation work at the Site. We evaluate the construction worker scenario quantitatively using modeled EPCs (see section 3.3.6). Chemical-specific risk estimates are not presented for these pathways, however the contribution of these pathways to total Site risk are shown in RAGS Part D Tables 7, 8, 9 and 10 and in Section 5.3 of this report.

3.2 Adequacy of Database for Calculating Exposure Point Concentrations

This section considers the adequacy of the COPC concentration database for estimating potential human exposure and risk at the Site. Site sampling largely targeted source areas rather than following a random or systematic sampling plan. As a result, EPCs based on these data provide a conservative representation of potential human exposure. A further degree of conservatism arises from use of the 95% upper confidence limit (UCL) of the mean as the EPC for Site contaminants. USEPA recommends use of the upper 95% UCL to account for uncertainty in the underlying database, including uncertainty associated with sample collection, analysis, and how well the data represent Site contamination that people are likely to contact. While people might be exposed to maximum Site concentrations at some point, the average is regarded as a reasonable estimate of concentration likely to be contacted over time. However, if the 95% UCL exceeds the maximum COPC concentration detected at the Site, the maximum concentration is used to represent the EPC.

In this assessment, we use Site data to estimate potential current and future human exposure at the Site. COPC concentrations vary in space and time, including seasonal variation. To the extent possible, this

variability was taken into account in the sampling design. Site data reflect current Site conditions, with all fully-validated samples collected in 1997 and 1998. It is reasonable to use these data to assess current exposure. We use current COPC concentrations to estimate future exposure, assuming steady-state conditions. In general, average concentrations in all media are likely to decrease over time; therefore, using current data to estimate future exposure is protective of human health. However, concentrations in nearby residential wells may increase over time as the plumes of contamination move, potentially impacting a larger geographic area.

The groundwater concentration data set is large, providing monitoring data over the last several years. Groundwater elevation can vary seasonally, and we assume it can vary by as much as ± 5 feet. Consequently, we use groundwater data as deep as 15 feet below ground surface in EPC calculations, compared to using soil data only from 0 to 10 feet below ground surface.

Surface water and sediment samples were collected in early October. At that time of year, contamination from spring runoff is not likely to be detected. Sediment samples were collected from depositional areas, where sediments, and COPCs, are likely to accumulate. Sampling from these areas is expected to provide a reasonable estimate of upper-end sediment concentrations.

Fish sampling covered the entire reach of Kelley Brook adjacent to the Site and beyond. USEPA Region I personnel believe that nearly all, if not all, fish present in the sampled reaches were caught (S. Svirsky, USEPA Region I, personal communication). Therefore, the data should be very representative of Kelley Brook fish. Stocked trout were the target of fish sampling, but only one stocked fish was caught. The rest of the fish were smaller native species. NHDES no longer stocks Kelley Brook.

Extensive soil sampling was conducted on Parcel 1, with less sampling on Parcel 2. If people spend more time in contact with portions of the Site that have higher levels of contamination than the rest of the Site, it may be necessary to assess risk in this area separately. For this reason, in addition to calculating the 95% UCL, we consider the potential for soil contamination "hot spots" in the discussion of soil EPCs in section 3.3.1.

3.3 Exposure Point Concentrations

Exposure point concentrations (EPCs) are concentrations of COPCs in various media to which receptors are exposed. They are defined by the exposure point, or location where a receptor may contact chemicals. Therefore, EPCs are media-specific and may differ depending on the exposure scenario. Most of these EPCs are based on measured data. All EPCs were calculated assuming that non-detected COPCs were present at ½ the detection limit.

We estimate both a reasonable maximum exposure (RME) and central tendency exposure (CT) pathway-specific EPC for all COPCs following USEPA Region 1 policy (1994, 1995). Most of the time, both the RME EPC and the CT EPC are represented by the 95% UCL of the mean. Groundwater is the major exception. We use maximum and average groundwater COPC concentrations to represent the RME EPC and the CT EPC, respectively. This approach is used given that residents could draw water from any single location in the future.

All COPC data sets with ten or more detected concentrations were tested to determine the concentration distribution shape. We used the Shapiro-Wilk test and Q-Q normal probability plots to determine if distributions follow normal or lognormal distributions (Appendix F). Most distributions are lognormally distributed. However, some distributions were not normal or lognormal. To simplify this task, we elected

to use the maximum COPC concentration as the RME EPC and the average COPC concentration as the CT EPC, as was done for groundwater data, for COPC that did not follow normal or lognormal distributions. If more detailed distributional analysis was performed for COPC concentration data that are not normally or lognorally distributed, EPCs for these COPCS would likely be lower than the maximum concentration used in this assessment. This same approach was used to establish RME EPCs and CT EPCs for COPC data sets with less than ten detected concentrations. We did not test the distribution shape of these distributions because we were not likely to obtain a good estimate of the 95% UCL (USEPA, 1992a; USEPA, 1997b).

Appendix H includes RAGS Part D Tables 3.1 to 3.16 that summarize pathway-specific EPCs for all COPCs at the Site. A separate table is included for each unique combination of scenario timeframe, medium, exposure medium, and exposure point. Assumptions used to calculate EPCs for each media are described in the following sections.

For some exposure pathways without measured data, we use screening models to estimate EPCs. Modeling was performed for trespasser, construction worker, and residential exposure to fugitive dust and soil vapors.

3.3.1 Soil EPCs

Sample results that appear to define the limits of Site contamination (i.e. no COPCs were detected in them) were excluded from soil EPC estimates. Soil contamination appears to be limited to Parcel 1, the soil piles, and those portions of Parcel 2 adjacent to the soil piles. Beyond the border with Parcel 1, COPCs were very rarely detected in Parcel 2 soil samples.

PCB Data

Aroclor data reveal a fairly consistent pattern of PCB contamination across the Site, with most PCBs quantified as Aroclor 1242 and occasionally as Aroclor 1260.

The PCB data set also includes PCBs quantified as homolog groups and individual congeners using gas chromatography/mass spectrometry (GC/MS) and as Aroclor mixtures. The GC/MS data should provide higher quality data than the Aroclor data, especially when PCB contamination is weathered and no longer strongly resembles the original Aroclor mixture. For this reason, we prefer to use the GC/MS data, summing across homolog groups, to calculate total PCB EPCs. However, the Aroclor data cover areas of the Site where PCBs were not analyzed using GC/MS and include some fairly high PCB concentrations (e.g., 270 mg/kg). Therefore, the Aroclor data were used with the homolog data to estimate EPCs for total PCBs.

The dioxin-like PCB congener concentrations were subtracted from the homolog data (but not the Aroclor data) prior to calculating the total PCB EPC for estimating cancer risk. The total PCB EPC for estimating non-cancer hazard includes these dioxin-like PCB congeners. Dioxin-like PCB congeners represent, on average, 7% of total PCB concentrations at the Site; therefore, subtracting them will not have a large effect on cancer risk estimates for PCBs.

Four soil samples were collected near Kelley Road (S-200, S-201, S-202, and S-203) and analyzed for PCBs to determine if Site contamination might impact adjacent residential properties. PCBs were very weathered in these samples and did not match the characteristic pattern of any Aroclor mixture. Therefore, the laboratory noted this result and reported the samples as nondetect for the six target Aroclors. However, the validator estimated J-qualified PCB concentrations in these samples of 150, 240,

840, and 1080 µg/kg quantified as Aroclor 1242, the Aroclor mixture most prevalent at the Site. These levels might reflect the influence of Site contamination and the nearby roadway.

Surface Soil (0 to 1 foot below ground surface)

Surface soil samples (i.e. 0 to 1 foot) on Parcel 1, three samples near the soil piles on Parcel 2 (S-204, S-205, S-206), and soil pile data were included in the current surface soil EPC. Other soil samples were excluded from the surface soil EPC calculation for the following reasons:

- ethylene glycol data were the only validated data available for samples S-54, S-55, S-56, and S-57.
- total organic carbon (TOC) data was the only validated data in samples SP-2, SP-7, SP-101, and SP-146/S-1.

Future Surface/ Subsurface Soil (0 to 10 feet below ground surface)

Surface (0 to 1 foot) and subsurface soil (1 to 10 feet below ground surface) samples from Parcel 1, Parcel 2 soil pile samples, and three samples near the soil piles on Parcel 2 (S-204, S-205, S-206) are combined to calculate the future soil EPC. Samples S-54, S-55, S-56, S-57, SP-2, SP-7, SP-101, and SP-146/S-1 were not included in the future soil EPC calculation for the reasons provided above in the description of the surface soil EPC. Samples S-207, S-22, S-23, and SP-26 are also near soil piles but not included in the EPC because they either were analyzed only for TOC, were analyzed with only screening-level analysis, or exhibited extremely low concentrations that likely define the limit of Site contamination.

Potential "Hot Spot Analysis"

The 95% UCL is an appropriate EPC for soil as long as contamination across the Site is relatively homogeneous. If Site contamination is heterogeneous and people spend more time in the more highly contaminated areas of the Site, it may be necessary to estimate potential risk just for this area. For example, averaging concentration data from this area into other Site data might "dilute" the EPC for people who build a home in the area of higher COPC concentrations. For this reason, we examined soil data to identify potential "hot spots" of contamination, loosely-defined as areas with extremely high COPC concentrations not well-represented by the 95% UCL of the mean.

Our hot spot analysis consisted of six steps:

1. Compare the maximum soil concentration of each COPC with its arithmetic average concentration in all other soil samples.
2. Flag those COPCs with maximum soil concentrations more than ten times higher than the arithmetic average concentration calculated from all other samples.
3. Consult COPC concentration distribution plots (Appendix E) and identify sample locations where the ten highest COPC concentrations were measured.
4. Use this information in conjunction with a Site map to identify potential "hot spot" exposure areas.
5. Calculate a 95% UCL on the mean for all COPCs for each potential "hot spot" exposure area and compare them.
6. Determine whether the 95% UCLs are substantially different, warranting a separate risk characterization for one or more of these potential "hot spots."

Table 3 summarizes the results from Steps 1 and 2. It identifies sampling locations with COPC concentrations that are ten times higher than the arithmetic average concentration for all other sampling locations and that also exceed USEPA Region III residential RBCs (corresponding to a cancer risk of 10E-6 and hazard quotient of 1). These results were considered in conjunction with the COPC distribution

plots to identify the potential exposure areas in Figure 6. Please note that Figure 6 delineates six of the ten potential exposure areas. The remaining 4 exposure areas are defined in Table 4, which also lists EPC estimates (95% UCL or maximum COPC concentration, whichever is lower) for each of these areas. Figure 7 graphically compares PCB and lead EPCs for each of these areas. With the exception of SWRP 1, which is represented by only three samples, these separate areas are all likely to be associated with significant levels of risk for these two COPCs. For this reason, it would not be particularly useful to calculate separate EPCs and risk estimates for each of these areas. Therefore, we calculated COPC EPCs using soil data from all of these areas.

While the "hot spot" analysis did not result in the calculation of risk for multiple soil exposure areas, it did reveal some important information about the spatial distribution of Site contamination. The distribution plots in particular illustrate the importance of sample S-223 (in the Tanks 1-21 exposure area), in which the highest lead concentration was measured. The part of the Site represented by this sample clearly poses unacceptable human health risk for the future residential exposure scenario.

The "hot spot" analysis also revealed that the four highest mercury concentrations were detected in soil piles 5A and 5B. The piles came from the original interceptor trench built just to the northwest of the demolished Site building. Significant human health risk might be associated with mercury levels in this pile, therefore, we used the 95% UCL from soil piles 5A and 5B as the EPC for mercury for the future resident exposed to soil at depth (0-10 ft soil). We used the 95% UCL from surface (0-1 ft) soil as the EPC for the future resident exposed to surface soil and for the current trespasser exposure scenario. High concentrations of other COPCs are generally correlated with one another, located primarily within obvious source areas (e.g., tank rows, lagoon, old Site building/former 140,000 gallon UST, truck storage area).

Table 3. Potential Soil Hot Spots

Sampling locations with contaminant concentrations that are ten times higher than the arithmetic average concentration for all other sampling locations and that exceed USEPA Region III residential RBCs (corresponding to a cancer risk of 10E-6 and hazard quotient of 1).

Sample Location	Sb	Ba	Cd	Cr	Cu	Pb	Hg	Ni	Mo	C_9-C_{18} aliphatics	Bis (2-ethylhexyl) phthalate	C_{11}-C_{22} aromatics
TP127/139	x		x		x							
TP145			x	x	x							
S-217	x											
P4-2					x							
P5A & P5B							x					
S-223		x	x	x		x		x	x			x
S-87											x	x
S-40									x			
S-214										x		
P7-1											x	
S-77											x	

Table 4. Exposure Point Concentrations for 10 Potential Soil Exposure Areas

Location:	Lagoon [a]	Old Site building and old UST/AST area [a]	SWRP 1 [a]	Tanks 1 - 21 [a]	Tanks 23 - 57 [a]	Loading dock area [a]	Rest of Parcel 1 [a]	All Parcel 1 Soils	Parcel 1 surface soil	Parcel 2 soils
	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]	EPC [b]
Inorganics (mg/kg)										
Antimony	5.80E-01	4.63E+00	2.70E-01	6.20E+00	5.60E-01	4.20E-01	6.78E-01	7.64E-01	7.06E-01	9.35E-01
Arsenic	1.21E+01	7.54E+00	5.70E+00	2.17E+01	9.90E+00	7.70E+00	6.83E+00	6.92E+00	7.04E+00	6.00E+00
Barium	9.05E+01	1.29E+02	5.44E+01	3.18E+03	7.18E+01	1.09E+02	7.17E+01	8.62E+01	1.06E+02	5.03E+01
Beryllium	7.75E-01	5.63E-01	2.80E-01	3.63E-01	3.80E-01	3.30E-01	4.30E-01	4.23E-01	3.57E-01	4.78E-01
Cadmium	7.30E-01	3.81E+00	5.50E-01	2.26E+01	4.80E-01	1.10E+00	1.18E+00	1.57E+00	1.90E+00	8.60E-01
Chromium	1.32E+01	4.48E+01	1.28E+01	5.28E+02	2.03E+01	2.84E+01	1.99E+01	2.51E+01	2.86E+01	2.22E+01
Copper	6.70E+00	5.26E+02	1.81E+01	2.53E+01	2.27E+01	4.12E+01	3.21E+01	4.92E+01	2.15E+01	6.41E+01
Lead	1.07E+03	1.19E+03	1.24E+02	1.99E+04	5.94E+02	4.53E+02	3.25E+02	6.19E+02	1.21E+03	1.47E+02
Manganese	1.14E+02	1.86E+02	7.40E+01	2.20E+02	1.02E+02	1.14E+02	2.08E+02	1.67E+02	1.29E+02	3.10E+02
Mercury	6.75E-02	2.42E+01	1.60E-01	2.00E+00	2.40E-01	4.40E-01	4.23E-01	1.16E+00	3.63E-01	6.12E-01
Nickel	1.37E+01	2.99E+01	9.20E+00	9.20E+02	1.52E+01	1.34E+01	1.49E+01	1.89E+01	2.40E+01	1.75E+01
Vanadium	1.10E+01	1.90E+01	1.22E+01	1.62E+01	1.71E+01	1.75E+01	2.00E+01	1.81E+01	1.57E+01	1.95E+01
Zinc	9.59E+01	4.79E+02	9.90E+01	1.58E+03	7.72E+01	1.35E+02	1.08E+02	1.39E+02	1.21E+02	1.11E+02
Molybdenum	1.62E+00	5.71E+00	2.00E+00	2.10E+01	1.90E+00	1.68E+01	1.89E+00	2.46E+00	4.22E+00	9.28E-01
VOCs (µg/kg)										
cis-1,2-Dichloroethene	1.10E+04	5.00E+00	ND	ND	ND	ND	ND	1.35E+02	4.00E+00	ND
Tetrachloroethene	5.80E+02	1.20E+01	ND	2.30E+03	1.20E+03	ND	3.20E+00	1.60E+02	1.69E+03	ND
Naphthalene	2.19E+04	8.60E+02	2.16E+03	5.50E+02	ND	2.50E+02	1.94E+02	7.57E+02	5.90E+02	1.36E+03
PAHs (µg/kg)										
Benzo(a)anthracene	1.60E+02	1.29E+03	1.60E+03	4.20E+03	6.30E+02	5.90E+02	1.12E+03	9.38E+02	4.20E+03	1.30E+03
Chrysene	3.20E+02	1.50E+03	1.00E+03	7.20E+03	8.10E+02	9.00E+02	8.37E+02	8.24E+02	2.76E+03	1.02E+03
Benzo(b)fluoranthene	6.70E+01	7.52E+02	6.00E+02	7.70E+02	4.10E+02	3.90E+02	7.33E+02	5.70E+02	1.45E+03	6.95E+02
Benzo(k)fluoranthene		6.96E+02	4.60E+02	4.80E+02	3.30E+02	3.50E+02	7.30E+02	5.43E+02	1.65E+03	8.95E+02
Benzo(a)pyrene	5.80E+01	9.25E+02	3.00E+02	7.10E+02	2.90E+02	4.30E+02	7.95E+02	6.09E+02	1.80E+03	9.20E+02
Indeno(1,2,3-cd)pyrene	2.20E+01	4.20E+02	2.40E+02	4.40E+02	2.80E+02	2.10E+02	5.14E+02	3.78E+02	9.62E+02	5.75E+02
Dibenz(a,h,)anthracene		1.15E+02	ND	2.70E+01	3.00E+01	ND	1.72E+02	2.16E+02	2.80E+02	1.20E+02
SVOCs by 8270 (µg/kg)										
bis(2-Ethylhexyl)phthalate	5.90E+04	4.22E+04	6.90E+04	1.30E+05	7.50E+04	3.40E+04	2.95E+04	4.71E+04	1.30E+05	1.32E+04
Extractable Petroleum Hydrocarbons (µg/kg)										
C9-C18 Aliphatics	1.43E+06	8.70E+05	4.58E+06	2.20E+06	1.69E+06	4.60E+05	5.96E+05	1.30E+06	4.58E+06	1.24E+06
C11-C22 Aromatics	2.64E+06	1.00E+06	2.71E+06	8.15E+06	2.02E+06	1.99E+06	2.27E+05	8.75E+05	8.15E+06	4.28E+05
Total PCBs (µg/kg)	8.89E+03	5.24E+04	5.50E+02	2.85E+05	6.67E+05	9.83E+04	3.26E+04	1.27E+05	4.88E+05	5.50E+04

[a] Exposure area represents a subset of Parcel 1 subsurface and surface soils.
[b] Exposure Point Concentration (EPC) is the 95% UCL on the mean or the maximum detected concentration if the 95% UCL is greater than the maximum.

3.3.2 Groundwater EPCs

In the future, off-Site and on-Site residents might be exposed to groundwater if they use it as a source of drinking water. Outdoor workers may contact groundwater during construction activities.

Drinking Water Exposure in the Future

Several groundwater samples were excluded from calculation of EPCs for residential exposure to tap water originating from groundwater at the Site. Samples AE-10, AE-20, MW-4, SH-30S, and SH-31S are excluded because they are described as background locations in SHA's draft RI (SHA, 1999, pg.109). Several samples were excluded because they appear to define the extent of Site plumes since COPCs were not detected in them (i.e. SH-27, SH-28, SH-29, WP-17, WP-18, SH-21D, SH-21I, SH-21S, SH-22D, SH-22S, SH-23D, SH-23I, SH-23S, SH-24D, SH-24I, SH-24S, SH-25D, SH-25I, SH-25S and SH-58S).

SH-24I contains nitrate-nitrogen at a concentration in excess of the NH AGQS of 10 mg/L. However, this well is not currently being used for tap water.

Several wells (AE-10, AE-21, AE-22, SH-32S, SH-33S, and SH-57S) were resampled because methylene chloride was detected in a trip blank in the first set of samples. O'Reilly, Okun, and Talbot reviewed both sets of data and determined that there was no analytical reason to eliminate the first set of data. In general the results agreed fairly well. Therefore, if a compound was detected in both sets of data, the results were averaged. If the compound was not detected, the lower detection limit was used as the EPC. If a compound was detected in one data set, but not in the other, the detected concentration was used as the EPC.

Several wells were sampled on multiple sampling dates. VOC data for these wells (AE-4, AE-10, AE-21, AE-22, SH-32S, SH-33S, and SH-53S) were averaged to calculate EPCs.

Current Drinking Water Exposure

People might be exposed to groundwater contamination at the Site through ongoing use of groundwater as a source of tap water. NHDES conducts periodic monitoring of residential wells to determine if any are being impacted by Site contamination. Some monitoring data for these wells are included in the preliminary draft RI report for the Site (SHA, 1999).

The current drinking water EPC is based on concentration data from two wells with point-of-entry treatment systems. The EPC is based on untreated groundwater samples from wells on the following lots: 51-1-1 and 32-3-4. The well on lot 51-1-1 serves a 12-unit condominium development. The well on lot 32-3-4 serves three dwellings. By incorporating only pre-treatment system data, these EPCs show what risk would be in the absence of the treatment systems.

Wells on the following lots also appear to be impacted by the Site: the residential well at 51-1-1, 33-8-13, 51-1-08, and possibly 32-2-05. NHDES continues to monitor nearby residential wells on a quarterly to annual basis and will install treatment systems if concentrations in excess of AGQSs are measured in other water supplies.

One residence (lot 32-3-11) adjacent to Parcel 1 used a shallow dug well in the basement as a drinking water supply until 1990 when petroleum-related contamination was discovered. This residence now uses a bedrock well on Parcel 1, near the former ethylene glycol recycling building. To date, Site-related contamination does not appear to be impacting this well.

Excavation Site

We assume that a construction worker will not be exposed to groundwater deeper than 15 feet below ground surface. This cutoff was selected assuming workers might dig to ten feet and that groundwater elevations might fluctuate by as much as ±5 feet. Therefore, we exclude data from all intermediate and deep groundwater wells (groundwater sampled at greater than 15 feet in all of these wells) and some shallow wells with samples collected at greater than 15 feet below ground surface. We also exclude wells in which all COPCs were never detected. Given these restrictions, the following wells are included in the future groundwater EPC for an excavation site: AE-3, AE-17S, BR-28, SH-6S, SH-15S, SH-20S, SH-41S, SH-42S, SH-43S, SH-45S, SH-48S, SH-49S, SH-50S, and SH-52S.

Vapor Intrusion to Indoor Spaces

Groundwater contaminant plumes across the Site are relatively deep. However, several COPCs detected in groundwater less than 15 feet below ground surface screen in using NHDES GW-2 standards (Appendix D, Table 2.10). Some, but not all, of these sampling locations are unlikely sites of future residential development given existing land use restrictions in wetlands and wetland buffer zones (100 feet). If we consider all groundwater data, the same COPC list emerges for this exposure pathway. See also discussion in Section 3.1.1 and 3.1.2.

3.3.3 Surface Water EPCs

Appendix G shows which surface water samples were used to calculate surface water EPCs. SW-1, SW-2, and SW-13 were excluded from the EPC calculation because they are located upstream of the Site.

3.3.4 Sediment EPCs

We used sediment samples collected in and immediately downstream of the area where free product historically discharged to Kelley Brook (from OS-5 to OS-10) to calculate sediment EPCs.

3.3.5 Fish EPCs

We used only brook trout data to calculate fish tissue EPCs. People in the area typically do not consume crayfish. People do eat pickerel, but cumulative consumption rates across all recreational fisher respondents to a 1991 survey are low (45 kg/yr) relative to brook trout consumption rates (420 kg/yr) for freshwater streams (USEPA 1997d, Table 10-66). Exclusion of pickerel data makes little difference in EPCs based on whole fish concentration data. Also, lipid data are available to estimate edible tissue concentrations for brook trout, but not for pickerel. For these reasons, we excluded pickerel data from the fish tissue EPCs.

Fish tissue EPCs were calculated using brook trout lipid content data to estimate edible tissue concentrations from whole body concentrations. Sidwell (1981) reported the lipid content for brook trout whole-body tissue as 5.2% (range=1.9-7.8). This number was divided by the lipid content of muscle tissue (1.6%, range=0.7-2.1), yielding a ratio of 0.31. While lipid content varies, we assume that muscle and whole body lipid content is correlated and that 0.31 is a reasonable estimate of the average ratio. Making this assumption, we adjusted whole body concentrations of lipophilic contaminants: PCBs, pesticides, and dioxins/furans.

Cooking can reduce the concentration of organic contaminants in fish (Sherer and Price, 1993; Wilson et al. 1998), but fish EPCs were not adjusted to account for this potential reduction in edible fish tissue concentration. We discuss this source of uncertainty further in the Risk Characterization.

3.3.6 Modeled EPCs

Air measurements were not made at the Site, therefore, we model concentrations in fugitive dust and vapors from soil using the particulate emission factor and volatilization factor described in USEPA's Soil Screening Guidance (SSG) (1996).

Fugitive dust

We calculate a particulate emission factor (PEF) to relate the concentration of COPCs in soil with the concentration of COPCs adhered to fugitive dust particles in outdoor air. To determine a fugitive dust EPC, we divide the soil EPC by the PEF. We calculate the PEF using the following equation:

$$PEF\left(\frac{m^3}{kg}\right)=\frac{Q}{C}*\frac{3600\,\text{sec}/hr}{0.036*(1-V)*\left(\frac{U_m}{U_t}\right)^3*F(x)}$$

Where:

Q/C	=	inverse of mean concentration at center of square source (g/m^2-s per kg/m^3)
V	=	fraction of vegetative cover (unitless)
U_m	=	mean annual windspeed (m/s)
U_t	=	equivalent threshold value of windspeed at 7 meters (m/s)
F(x)	=	function dependent on Um/Ut derived using Cowher et al. (1985) (unitless)

We use a value of 71.87 for the Q/C parameter, which is the value from Table 3 of the SSG for a 1 acre source in Harrisburg, PA. We assume zero vegetative cover because a large part of Parcel 1 is bare soil without grasses or trees. We used EPA defaults for U_m, U_t and F(x). The values and sources used to calculate the PEF are presented in Table I-1 in Appendix I. These values are intended to provide a conservative, screening level estimate of exposure to fugitive dust.

Volatilization from soil

We calculate a volatilization factor (VF) to define the relationship between the concentration of COPCs in soil and the flux of volatile contaminants to outdoor air. To calculate a soil vapor EPC, we divide the soil EPC by the VF. We calculate the VF using the following equation:

$$VF\left(\frac{m^3}{kg}\right)=\frac{Q}{C}*\frac{(3.14*D_A*T)^{1/2}}{2*\rho_b*D_A}*10^{-4}\left(\frac{m^2}{cm^2}\right)$$

Where:

Q/C	=	inverse of mean concentration at center of square source (g/m^2-s per kg/m^3)
D_A	=	apparent diffusivity (cm^2/s)
T	=	exposure interval (s)
ρ_b	=	dry soil bulk density (g/cm^3)

We use a value of 71.87 for the Q/C parameter, which is the value from Table 3 of the SSG for a 1 acre source in Harrisburg, PA. The values and sources used to calculate the VF are presented in Table I-2 in Appendix I. These values are intended to provide a conservative, screening level estimate of exposure to soil vapor.

3.4 Estimating Average Daily Doses

To estimate average daily doses received by people exposed to Site contamination, one must combine EPCs with variables that describe contact rates with Site media (e.g., soil ingestion rate, type of outdoor

activities), physiological data of receptors (e.g., body weight, inhalation rate), and time-activity pattern data (e.g., swimming frequency and duration). Appendix J includes RAGS Part D Tables 4.1 through 4.28, which describe RME and CT exposure assumptions for each unique combination of scenario timeframe, exposure medium, exposure point, receptor population, and receptor age. Appendix I presents model parameters for modeling fugitive dust and vapors from soil.

RME and CT exposure assumptions are derived primarily from USEPA Region I guidance and the USEPA Exposure Factors Handbook (1997c, d, e). Dermal absorption efficiencies were selected from the primary literature and USEPA dermal exposure guidance (USEPA, 1992). RME and CT exposure assumptions are combined with EPCs to estimate daily COPC intakes for each exposure route and exposure point using the following general equation:

$$ADD = \frac{Total\ Amount\ of\ Contaminant\ Intake}{(Body\ Weight_{average})\ (Averaging\ Period)} \times Absorption\ Factor$$

The RME average daily dose is the highest exposure that is reasonably expected to occur at the Site. The CT average daily dose provides an average exposure estimate. Together, these two estimates convey a range of potential exposures at the Site.

Two ADDs are calculated for each exposure route: the ADD(year) and the ADD(life). The ADD(year) is used to evaluate non-carcinogenic effects. It represents the chemical dose during the exposure period and is calculated as the average daily dose over an appropriate averaging period. The ADD(life) is used to evaluate carcinogenic effects. It represents the chemical dose averaged over a lifetime and is calculated as the average daily dose over a 70-year lifetime.

Duration of the averaging period is significant because different effects may be manifested at different dose levels, and over different durations. The averaging period is important for effects for which there may be thresholds. Thresholds are defined as the dose below which deleterious effects are not likely for even the most sensitive populations. Probable carcinogens are not considered to have thresholds because any exposure is assumed to present some risk.

The equations used to estimate the Average Daily Dose of chemicals via several exposure routes are shown in Sections 3.4.1 through 3.4. 8.

3.4.1 Ingestion of Chemicals in Drinking Water

$$ADD(mg/kg-day) = \frac{EPC_W \times IR \times EF \times ED}{BW \times AT}$$

where:

EPC_w	=	Chemical concentration in water (mg/liter)
IR	=	Ingestion rate (liters/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)

BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged: days)

3.4.2 Incidental Ingestion of Chemicals in Surface Water While Wading

$$ADD(mg/kg-day) = \frac{EPC_W \times CR \times ET \times EF \times ED}{BW \times AT}$$

where:

EPC_w = Chemical concentration in water (mg/liter)
CR = Contact rate (liters/hour)
ET = Exposure time (hours/event)
EF = Exposure frequency (events/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged: days)

3.4.3 Dermal Contact with Chemicals in Water

$$ADD(mg/kg-day) = \frac{EPC_W \times SA \times PC\,x\,ET \times EF \times ED\,x\,CF_w}{BW \times AT}$$

where:

EPC_w = Chemical concentration in water (mg/liter)
SA = Skin surface area available for contact (cm^2)
PC = Chemical-specific dermal permeability constant (cm/hour)
ET = Exposure time (hours/day)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
CF_w = Volumetric conversion factor for water (1liter/1,000cm^3)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged : days)

3.4.4 Ingestion of Chemicals in Soil (and Sediment)

$$ADD(mg/kg-day) = \frac{EPC_S \times IR \times CF \times FI \times EF \times ED}{BW \times AT}$$

where:

EPC_s = Chemical concentration in soil (or sediment) (mg/kg)
IR = Ingestion rate (mg soil or sediment/day)
CF = Conversion factor (10^{-6} kg/mg)
FI = Fraction ingested from contaminated source (unitless)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)

BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged: days)

3.4.5 Dermal Contact with Chemicals in Soil (and Sediment)

$$ADD(mg/kg-day) = \frac{EPC_s \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

where:

EPC_s = Chemical concentration in soil (or sediment) (mg/kg)
CF = Conversion factor (10^{-6} kg/mg)
SA = Skin surface area available for contact (cm^2/event)
AF = Soil to skin adherence factor (mg/cm^2)
ABS = Absorption factor (unitless)
EF = Exposure frequency (events/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged: days)

3.4.6 Inhalation of Airborne (Vapor Phase) Chemicals

$$ADD(mg/kg-day) = \frac{CA \times IR_a \times ET \times EF \times ED}{BW \times AT}$$

where:

CA = Contaminant concentration in air (mg/m^3)
IR_a = Inhalation rate (m^3/hour)
ET = Exposure time (hours/day)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged: days)

3.4.7 Inhalation of Chemicals Adsorbed to Fugitive Dust

$$ADD(mg/kg-day) = \frac{\left(\frac{EPC_s}{PEF}\right) \times IR_a \times ET \times EF \times ED}{BW \times AT}$$

where:

EPC_s = Chemical concentration in soil (mg/kg)
PEF = Particulate Emission Factor (m^3/kg)
IR_a = Inhalation rate (m^3/hour)
ET = Exposure time (hours/day)
EF = Exposure frequency (days/year)
ED = Exposure duration (years)

BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged : days)

3.4.8 Ingestion of Contaminated Food

$$ADD(mg/kg-day) = \frac{EPC_f \times IR \times FI \times EF \times ED}{BW \times AT}$$

where:

EPC_f = Contaminant concentration in food (mg/kg)
IR = Ingestion rate (kg/meal)
FI = Fraction ingested from contaminated source (unitless)
EF = Exposure frequency (meals/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (period over which exposure is averaged : days)

4.0 DOSE-RESPONSE ASSESSMENT

To quantify risk, one must understand the relationship between the dose received and the incidence of an adverse effect. This relationship is often called the "dose-response relationship." For carcinogens, it is expressed as a cancer slope factor (CSF) or unit risk (UR). These values measure a carcinogen's potency via the oral and inhalation routes of exposure, respectively. For noncarcinogens, toxicity benchmarks are called Reference Doses (RfDs) and inhalation Reference Concentrations (RfCs).

This section provides the following information:

- Description of COPC toxicity values (i.e. RfDs, RfCs, CSFs, and URs);
- Adjustments to toxicity values for this assessment;
- Surrogate toxicity values for COPCs that lack toxicity values; and
- Approaches for estimating risk from exposure to PCBs, carcinogenic PAHs, petroleum fractions, and lead.

Appendix C provides brief profiles for each COPC that describe potential exposures to the compound, its physical and chemical properties, and a summary of toxicity information.

4.1 Available Toxicity Information

The toxicity assessment for compounds consists of two steps: (1) determining whether it results in observed toxic effects in animals or humans, and (2) identifying the dose-response relationship. The toxicity assessment considers a compound's potential to cause both cancer and non-cancer effects.

Quantitative estimates of a compound's toxicity are referred to as toxicity values. RfDs are average daily doses and RfCs are average daily concentrations of compounds below which adverse non-cancer health effects are not expected to occur. CSFs and inhalation URs are quantitative estimates of a compound's cancer potency. These toxicity values were selected from the following USEPA sources:

- Integrated Risk Information System database (IRIS, http://www.epa.gov/iris);
- National Center for Environmental Assessment (NCEA), Superfund Technical Support Center and on-line Toxicological Profiles (http://www.epa.gov/ncea); and,
- Health Effects Assessment Summary Tables (HEAST, USEPA 1997).

IRIS values receive the highest level of peer review; therefore, these values are preferred in quantitative risk assessment. If IRIS toxicity values are not available, provisional values from NCEA are used, followed by values from HEAST.

Tables 5.1 and 5.2 in Appendix K list RfDs and RfCs for COPCs. Tables 6.1 and 6.2 in Appendix L list CSFs and URs for carcinogenic COPCs. These tables indicate the source of toxicity values, assumptions made about the toxicity of COPCs with no published toxicity values, and any proposed values currently under consideration for inclusion in IRIS.

4.1.1 Evaluation of Non-cancer Health Effects Using RfDs and RfCs

RfDs and RfCs are used to evaluate the potential non-cancer effects of compounds exhibiting systemic toxicity via oral, dermal, and inhalation routes of exposure. RfDs for oral exposure must be adjusted to

represent the toxicity via the dermal exposure route**.** Exposure to these compounds must overwhelm organic homeostatic, compensating, and adaptive mechanisms before a toxic endpoint can occur. Thus, RfDs and RfCs are benchmarks designed to fall at or below the lowest threshold for toxic effects among the population to be protected.

USEPA formally defines RfDs and RfCs as follows:

- *RfDs (mg/kg-d)* are estimates (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious ***non-cancer*** effects during a lifetime; and
- *RfCs (mg/m^3)* are estimates (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious ***non-cancer*** effects during a lifetime.

Interaction of Exposure Duration and Health Outcomes

The assessment of non-carcinogenic effects is complicated by the interaction of time scales of exposure with types of effects (acute, subchronic, and chronic). Subchronic and chronic health effects are those that might occur following long term exposures typically of concern at hazardous waste sites. USEPA defines subchronic exposures as those lasting up to seven years. Chronic exposures are defined as those lasting more than seven years. Most available RfDs and RfCs are applicable to the evaluation of chronic rather than subchronic exposures. Chronic RfDs are used to evaluate subchronic exposures when a subchronic value is not available from IRIS, NCEA or HEASTE. The child resident is only 0- 6 yrs old, which is less than the typical chronic period of 7+ years. However, chronic toxicity criteria are used to evaluate risk to the child since the child simply represents one time period in the life of a person living for 30 years at the Site.

Subchronic toxicity values have been estimated for only a few COPCs; therefore, non-cancer hazard for the construction worker is based primarily on use of chronic RfDs and RfCs.

Derivation of RfDs and RfCs

RfD and RfC derivations start with the highest "no observed adverse effect level" (NOAEL), which is the dose or concentration at which there are no statistically or biologically significant increases in the frequency or severity of any effect between the exposed population and its appropriate control. Lowest observed adverse effect levels (LOAELs) are sometimes used when NOAELs are not available. Uncertainty factors are applied to NOAELs to ensure that RfDs and RfCs are sufficiently protective given uncertainties in the underlying toxicity database. Uncertainty factors (UF) are incorporated as divisors to the NOAEL associated with the critical effect (i.e. the first adverse effect, or its known precursor, that occurs as the dose rate increases). Standard uncertainty factors include:

- 10-fold factor for extrapolation from animals to humans;
- 10-fold factor for variability in the human population;
- 3 to 10-fold factor for use of a less-than-chronic study;
- 1 to 10-fold factor for extrapolation from a LOAEL to a NOAEL; and
- 3 to 10-fold factor for an incomplete database.

Application of these uncertainty factors results in RfDs and RfCs between 1 and 10,000 times lower than the NOAEL. An additional divisor, or modifying factor (MF), between 1 and 10 can be used to account for scientific uncertainties of the study and database not explicitly treated with the standard uncertainty factors. The default value for the MF is 1.

The use of these ten-fold uncertainty factors originated more than forty years ago (Lehman and Fitzhugh, 1954). However, some analysis of toxicity information provides theoretical, and sometimes experimental, support for their selection (Dourson and Stara, 1983). While these uncertainty factors appear to be protective for the "average" compound, toxicologists are beginning to develop more accurate uncertainty factors using the expanding knowledge of inter- and intra-species differences in sensitivity, mechanisms of toxicity, and a growing toxicological study database (Dourson et al. 1996). Some researchers are developing probabilistic characterizations of RfDs to explicitly account for uncertainty and variability inherent in these values (Baird et al. 1996).

Interpretation of RfDs and RfCs

Adverse effects are not likely at doses and concentrations below toxicity values. The level of concern for a particular COPC does not increase linearly as the RfD and RfC are approached or exceeded because these values are not equally accurate or precise, nor are they based on the same severity of toxic effects. In fact, the slopes of dose-response curves in excess of RfDs and RfCs can vary considerably among COPCs. Therefore, comparing these values with exposure estimates at the Site provides an index of concern rather than a probability of an adverse effect occurring.

4.1.2 Evaluation of Cancer Risk Using CSFs and URs

Carcinogenic potential is described by CSFs with units of $(mg/kg\text{-}day)^{-1}$ and URs with units of $(\mu g/m^3)^{-1}$. These values provide a quantitative estimate of the carcinogenic potency of chemicals to humans. Carcinogens can evoke changes in a single cell leading to uncontrolled cell proliferation. Theoretically, there is no level of exposure that does not pose a small, but finite, probability of causing cancer. Therefore, unlike systemic toxicants, carcinogens are assumed to have no threshold below which there is zero cancer risk.

According to USEPA Risk Assessment Guidelines of 1986, human carcinogenic potential is classified through a weight-of-evidence classification scheme (A through E), which provides information on the type and quantity of data available. USEPA's Proposed Guidelines for Carcinogenic Risk (1996) classify human carcinogenic potential as "known/likely", "cannot be determined", and "not likely", to replace the alphanumeric categories A-E. Tables 6.1 and 6.2 in Appendix L indicate the weight-of-evidence classification for all COPCs under the 1986 guidelines and, when available, under the proposed guidelines.

The weight-of-evidence evaluation considers available test data, adequacy of studies, types of studies, and observed responses. Chemicals that give rise to cancer or gene mutations are generally classified as follows:

- Group A: Human Carcinogen, sufficient human data;
- Group B1: Probable Human Carcinogen, limited human data;
- Group B2: Probable Human Carcinogen, sufficient evidence in animals and limited evidence or no evidence in humans;
- Group C: Possible Human Carcinogen, limited evidence in animals and limited or no evidence in humans;
- Group D: Not Classifiable as to Human Carcinogenicity, insufficient tests for carcinogenesis or mutagenesis are available; and
- Group E: Evidence of Non-Carcinogenicity in Humans.

The CSF or UR, as calculated in accordance with the USEPA 1986 guidelines, is usually the 95% statistical upper bound on the slope of the dose-response curve in the low-dose linear portion as estimated by the linearized multistage model (LMS). The larger the CSF or UR, the greater the cancer potency of the compound. In addition, in accordance with the USEPA 1986 guidelines, CSFs or URs are calculated assuming there are no threshold levels for carcinogenic effects and that the response increases linearly with dose at low levels, including dose levels encountered in the environment.

In the proposed guidelines for carcinogen risk assessment (USEPA 1996a), the compound's mode of action is emphasized. For carcinogenic agents whose mode of action is believed to influence later stages in the carcinogenesis process, a threshold is believed to exist. For these compounds, a certain level of the compound is required in the cellular population before it can influence cancer formation. Such thresholds are not considered for carcinogens in the 1986 guidelines. Another variation from the 1986 guidelines is the use of straight-line extrapolation for non-threshold carcinogens rather than the LMS. When available, CSFs and URs developed in accordance with USEPA's proposed cancer guidelines (1996) are presented in Tables 6.1 and 6.2. However, assessments based on the 1986 guidelines are considered scientifically acceptable for estimating human health risk.

4.2 Conversion of Inhalation Toxicity Concentrations (RfC and URs) to Inhalation Toxicity Doses (RfDs and CSFs)

RfCs are converted to inhalation RfDs by multiplying the RfC by the inhalation rate of 20 m^3/day assumed in deriving the RfC and dividing by a body weight of 70 kg. The equation for adjusting RfCs for each $COPC_i$ is as follows:

$$RfD_i \text{ (mg/kg-day)} = RfC_i \text{ (mg/m}^3\text{)} \times 20 \text{ m}^3\text{/day} \times [1/70 \text{ (kg)}]$$

URs are converted to inhalation CSFs by multiplying by a body weight of 70 kg and dividing by an inhalation rate of 20 m^3/day. The equation for adjusting URs for each $COPC_i$ is as follows:

$$CSF_i \text{ (mg/kg-day)}^{-1} = \text{Inhalation UR } (\mu\text{g/m}^3)^{-1} \times 70 \text{ kg} \times [1/20 \text{ (m}^3\text{/day)}] \times 1000 \ \mu\text{g/mg}$$

4.3 Adjusting Oral Toxicity Values for Estimating Toxicity via the Dermal Route of Exposure

Most RfDs and CSFs are based on administered doses. The administered dose in a toxicity study is the amount of a compound given to an organism and in contact with an exchange boundary (e.g., gastrointestinal tract). ADDs calculated in this assessment for the ingestion pathway also represent administered doses and are comparable to COPC RfDs and CSFs.

ADDs calculated for the dermal exposure pathway represent absorbed doses rather than administered doses. Absorbed dose is the amount of a COPC penetrating the exchange boundaries of an organism after contact. The ADD equation for the dermal exposure pathway includes a chemical-specific absorption efficiency factor (Table 4.a) that accounts for the amount of COPC that permeates the skin and is absorbed by the body. Therefore, RfDs based on administered doses must be adjusted to represent absorbed doses before comparing to dermal ADDs. This adjustment consists of multiplying the oral RfD by the chemical-specific absorption efficiency in the gastrointestinal tract to obtain the fraction of the administered dose that is absorbed.

The oral to dermal RfD adjustment factors are listed in Tables 5.1 and 6.1, with references for each factor listed in Table 5.1a in Appendix K. These adjustment factors are particularly important for evaluating exposures to metals, because many metals are not well absorbed in the gastrointestinal tract. Without this adjustment, non-cancer hazard and cancer risk from dermal exposure to metals would be underestimated.

4.4 COPCs with No Published Toxicity Values

Some COPCs can be toxic to humans but have an inadequate toxicity database to support the derivation of toxicity values. In this assessment, we assign surrogate toxicity values wherever reasonable based on knowledge of the COPC's mechanism(s) of toxicity. This approach introduces uncertainty into the analysis but is judged to be more appropriate than ignoring these compounds.

4.4.1 VOCs

EPA recently classified chloroethane as a B2 probable human carcinogen via the inhalation route of exposure (NCEA, 1998). However, available studies do not provide adequate dose-response data to estimate the CSF. No surrogate CSF is assigned to this COPC. Non-cancer hazard associated with chloroethane was quantified.

No RfDs or RfCs are available for several alkylbenzenes at the Site:

n-butylbenzene	n-propylbenzene
sec-butylbenzene	1,2,4-trimethylbenzene
tert-butylbenzene	1,3,5-trimethylbenzene
p-isopropyltoluene	

However, these compounds are included in the VPH and EPH fractions measured at the Site; therefore, any non-cancer hazard they pose is considered in COPC screening of these fractions. Non-cancer hazard for fractions retained as COPCs are quantified in this assessment.

Several groundwater COPCs have no RfDs or RfCs: 1,2-Dichloroethane, 1,1,2,2-tetrachloroethane, and trichloroethene. However, these compounds were evaluated as carcinogens.

Non-cancer hazard and cancer risk from exposure to MTBE in Site groundwater was not estimated. Much of the available toxicity literature for MTBE comes from inhalation studies, with adequate data to derive an inhalation RfC. However, there is no oral RfD for MTBE; therefore, no non-cancer hazard from oral exposure could be quantified. We did not perform a cross-route extrapolation of the RfC, relying on USEPA's conclusion that "[t]he toxicokinetic models are … limited in helping to perform an adequate extrapolation from the inhalation data to actual oral exposures from drinking water intake" (USEPA 1997g).

MTBE

MTBE non-cancer hazard via the inhalation route of exposure can be quantified. However, inhalation risk from future use of Site groundwater as residential tap water is assessed qualitatively rather than quantitatively per USEPA Region I policy (1995). At this time, there is no CSF established for MTBE, so no cancer risk estimate was calculated.

Given that possible non-cancer hazard and cancer risk from MTBE exposure was not quantified at the Site, is there a reason to believe this COPC in groundwater might pose unacceptable risk to a future

resident? The maximum groundwater concentration of MTBE is 120 µg/L (Sample WP-3). The average concentration in all Site groundwater, excluding sample locations where MTBE was not detected, is 20 µg/L.

The USEPA recently prepared a drinking water advisory for MTBE (USEPA 1997g). In this advisory, USEPA recommends 20 to 40 µg/L in drinking water to avoid taste and odor problems, concluding that these concentrations are four to five orders of magnitude lower than concentrations associated with observed cancer and non-cancer effects in animals. In reaching this conclusion, USEPA acknowledged that there are "many uncertainties and limitations associated with the toxicity database for this chemical." NHDHHS recently adopted a primary drinking water standard for MTBE based on cancer risk of 13 µg/L, modifying the current standard of 70 µg/L.

Consequently, MTBE groundwater concentrations at the Site are a concern at least for odor and taste problems, if not health risk.

4.4.2 Pesticides

Cis-nonachlor and trans-nonachlor are COPCs in fish tissue, but these compounds have no toxicity values. Both compounds can be present in technical chlordane mixtures. Chlordane was detected in fish, but was not retained as a COPC because it was detected below the risk-based concentration (RBC) for human fish consumption developed by USEPA Region III (1999). Cis- and trans-nonachlor were retained as COPCs because they have no toxicity values and are present above background concentrations. We could substitute toxicity values of chlordane for these compounds, but there would be a great deal of uncertainty associated with this approach. Instead, these compounds are evaluated qualitatively in this assessment.

The RfD and UR for 4,4'-DDT are assigned to 4,4'-DDE, which has no published UR. DDT and DDE have the same oral slope factor, and the unit risk for 4,4'-DDT is simply derived from this oral slope factor.

4.4.3 PAHs

For this assessment, toxicity values for naphthalene were selected to represent the toxicity of 2-methylnaphthalene. The RfD for pyrene was used to represent the toxicity of acenaphthalene, benzo(g,h,i)perylene, and phenanthrene. Non-cancer hazard associated with other PAH COPCs (ie. carcinogenic PAHs) is not quantified. Cancer risk associated with seven carcinogenic PAHs is evaluated as described in Section 4.6.

4.5 Evaluation of Risk Associated with PCB Exposure

The potential cancer and non-cancer health effects of PCBs have been reviewed extensively (Silberhorn, 1990; Safe, 1994; Swanson et al., 1995; Longnecker et al., 1997; Rice, 1997; Jacobson and Jacobson, 1997; Cogliano, 1998; Geisy and Kannan, 1998; ATSDR, 1999). USEPA classifies PCBs as probable human carcinogens ("B2") based on animal toxicity data. Several congeners appear to have dioxin-like activity and are discussed in Section 4.5.3.

4.5.1 Non-Cancer Hazard - Total PCBs

The potential for non-cancer effects from PCB exposure is estimated using the RfD for Aroclor 1254. There is a slightly higher RfD available for Aroclor 1016, which is a less chlorinated mixture of PCB congeners than Aroclor 1254 (See Figure 8). However, the more conservative Aroclor 1254 RfD was used because PCB mixtures at the Site are typically more highly chlorinated than Aroclor 1016. Figures 8 and 9 compare congener patterns in Aroclor 1016 and Aroclor 1254 with congener patterns in Site soils (0-1 ft and 0-10 ft), Kelley Brook sediment, and brook trout caught in Kelley Brook.

4.5.2 Cancer Risk – Total PCBs Excluding TCDD-like Congeners

USEPA recommends a tiered approach to assess cancer risk associated with exposure to PCBs. Studies to date suggest that more highly chlorinated, less volatile congeners are associated with greater cancer risk. These congeners tend to persist in the environment in soils and sediment and bioaccumulate in biota.

When congener data are not available, the exposure pathway can be used to indicate how the potency of a mixture might have changed following release to the environment. For example, more volatile, less chlorinated congeners are more likely to be metabolized and eliminated than highly chlorinated congeners that persist in environmental media and bioaccumulate in biota. Therefore, a higher CSF (upper-bound estimate = 2.0 per mg/kg/day; central estimate = 1.0 per mg/kg/day) is used to evaluate risk from exposure to highly chlorinated congeners or exposure via pathways that tend to involve highly chlorinated congeners. This higher slope factor is used under the following conditions:

- Food chain exposure
- Sediment or soil ingestion
- Dust or aerosol inhalation
- Dermal exposure, if an absorption factor has been applied
- Presence of dioxin-like, tumor-promoting, or persistent congeners
- Early-life exposure (all pathways and mixtures)

Therefore, this CSF was used to estimate PCB cancer risk associated with exposure to soil, sediment, and fish.

A lower CSF (upper-bound estimate = 0.4 per mg/kg/day, central estimate = 0.3 per mg/kg/day) is used for more volatile PCB congener mixtures that are less persistent. This lower slope factor is used under the following conditions:

- Ingestion of water-soluble congeners
- Inhalation of evaporated congeners
- Dermal exposure, if no absorption factor has been applied

If congener or isomer analyses verify that congeners with more than 4 chlorines comprise less than 1/2% of total PCBs, USEPA recommends use of an even lower CSF (upper-bound estimate = 0.07 per mg/kg/day, central estimate = 0.04 per mg/kg/day). However, Site data do not meet this requirement.

4.5.3 Cancer Risk: TCDD-like PCB Congeners and Dioxin Congeners

2,3,7,8-TCDD is the most potent of a group of compounds that bind to an intracellular protein called the aryl hydrocarbon receptor (AhR). Some PCB congeners and dioxin congeners also bind to this receptor

and have been shown to exert toxic responses similar to those exerted by TCDD. The biological activity of these compounds seems to correlate with their binding affinity to this receptor. Toxic responses associated with binding to this receptor include developmental and reproductive toxicity, liver toxicity, immunotoxicity, and probably cancer (WHO 1998).

A toxic equivalency quotient (TEQ) approach has been developed to represent the fractional cancer and non-cancer toxicity of PCB and dioxin congeners relative to TCDD for congeners exhibiting the following characteristics: (1) structural relationship to PCDDs and PCDFs; (2) bind to the Ah receptor; (3) elicit AhR-mediated biochemical and toxic responses; and (4) are persistent and accumulate in the food chain. The TEQ approach assumes additivity among congener effects, but the predictive value of TEQs may be species- and response-dependent because both additive and antagonistic interactions have been observed (Safe 1999, Birnbaum 1999). Despite this uncertainty, use of the TEQ approach is appropriate given that consideration of TCDD alone might underestimate risk.

TEQs are calculated as follows:

$$TEQ = \Sigma\,[\text{TCDD-like Congener}_i \cdot TEF_i]_n$$

where,
TEF = toxic equivalency factor for congener i,
n = number of TCDD-like congeners in mixture of concern.

Toxic equivalency factors (TEFs) for each dioxin-like congener have been published recently (Van den Berg et al. 1998:

Dioxin Congeners	TEF	Dioxin-like PCB Congeners	TEF
2,3,7,8-TCDD	1	PCB-77	0.0001
1,2,3,7,8-PeCDD	1	PCB-81	0.0001
1,2,3,4,7,8-HxCDD	0.1	PCB-118	0.0001
1,2,3,6,7,8-HxCDD	0.1	PCB-123	0.0001
1,2,3,7,8,9-HxCDD	0.1	PCB-105	0.0001
1,2,3,4,6,7,8-HpCDD	0.01	PCB-114	0.0005
OCDD	0.0001	PCB-126	0.1
2,3,7,8-TCDF	0.1	PCB-128/167	0.00001
1,2,3,7,8-PeCDF	0.05	PCB-156	0.0005
2,3,4,7,8-PeCDF	0.5	PCB-157	0.0005
1,2,3,4,7,8-HxCDF	0.1	PCB-169	0.01
1,2,3,6,7,8-HxCDF	0.1	PCB-189	0.0001
1,2,3,7,8,9-HxCDF	0.1		
2,3,4,6,7,8-HxCDF	0.1		
1,2,3,4,6,7,8-HpCDF	0.01		
1,2,3,4,7,8,9-HpCDF	0.01		
OCDF	0.0001		

In Tables 6.1 and 6.2, the TCDD CSF is multiplied by the TEF for each TCDD-like congener to show the relative carcinogenicity of each congener. However, the TEFs are usually used as shown in the equation above, adjusting the congener's EPC downward to a TCDD-equivalent concentration rather than adjusting the CSF.

Combined cancer risk and non-cancer hazard associated with TCDD-like TEQ is evaluated by comparing the TEQ for each exposure scenario at the Site with the World Health Organization's (WHO) Tolerable Daily Intake (TDI) of 1-4 pg/kg-d. The TDI is intended to represent a tolerable daily intake for lifetime exposure with no adverse health consequences. General populations in industrialized nations now take in 2-6 TEQ pg/kg-d. At the WHO consultation where the TDI was recommended, participants stressed that the 4 pg/kg-d should be considered the maximum TDI on a provisional basis and that intakes below 1 pg/kg-d should be the future goal.

4.6 Application of Relative Potency Factors for Carcinogenic PAHs

USEPA classifies benzo(a)pyrene (B(a)P) as a Group B2, or probable human carcinogen. The oral cancer slope factor is based on a dietary study in mice published by Neal and Rigdon (1967). The data were modeled by two procedures to provide three upper bound estimates. A linearized multistage procedure was applied to data by Brune et al. (1981) to provide the fourth estimate. The range is 4.5 to 11.7 $(mg/kg\text{-}day)^{-1}$, with a median of 6.3 $(mg/kg\text{-}day)^{-1}$. The geometric mean of the four risk estimates is 7.3 $(mg/kg\text{-}day)^{-1}$.

Six other PAHs are classified as B2 carcinogens. Each of these PAHs generates biologically active metabolites associated with tumor formation. Results are consistent among cancer bioassays involving B(a)P and these PAHs; however, insufficient data are available to derive CSFs for all of these PAHs. Also, while these PAHs cause cancer by the same mechanism as B(a)P, they appear to be less potent. USEPA developed a relative potency approach to estimate cancer risk associated with these PAHs (USEPA 1993). This approach assumes that these PAHs have dose-response curves similar to that of B(a)P, but that it takes a proportionally larger concentration of these compounds to induce an equivalent tumor response. To develop relative potency factors, USEPA compared PAH relative cancer potencies within and across available cancer bioassays. These relative potency factors (RPFs) are used to assess only PAH cancer risk:

PAH	Relative Potency Factor (RPF)
Benzo(a)pyrene	1
Benz[a]anthracene	0.1
Benzo[b]fluoranthene	0.1
Benzo[k]fluoranthene	0.01
Chrysene	0.001
Dibenzo[a,h]anthracene	1
Indeno[1,2,3-cd]pyrene	0.1
Source: USEPA, 1993	

Similar to the TCDD-like congener TEQ approach described in Section 4.5.3, this approach assumes additivity of toxicity among the PAHs. However, both additive and nonadditive effects have been observed for the carcinogenicity and genotoxicity of PAHs by different exposure routes (USEPA 1993).

There is a provisional UR for quantifying cancer risk associated with inhalation exposure to B(a)P. It is not known whether PAHs are equipotent by the oral and inhalation routes, but we applied the RPFs to the provisional UR to estimate cancer risk from these PAHs.

4.7 Evaluation of Risk Associated with Lead Exposure

The disposition of lead is fairly well understood, as are the target organs, effects, and to some extent, the mechanism by which lead exerts its adverse effects. Although lead has been shown to affect every system in the body, the most sensitive target organs are the nervous system in young children, the hematopoietic system, and the cardiovascular system. The nervous system is by far the most sensitive target organ. Based on animal and human studies, it does not appear as though there is a threshold for the adverse effects of lead on this system.

USEPA classifies inorganic lead as a category B2, probable human carcinogen. There is inadequate evidence of carcinogenicity based on human studies, but several animal bioassays have shown statistically significant increases in renal tumors following dietary and drinking water exposure to lead acetate or lead subacetate, two soluble lead salts (IRIS, 1999b). No CSF is available for inorganic lead because of the large uncertainties involved, including the effect of age, health, nutritional status, and body burden (IRIS, 1999b).

USEPA has not established an RfD for inorganic lead because it appears that some observed effects occur at such low doses as to be essentially without a threshold (IRIS, 1999b). Alternatively, information on the distribution, metabolism, and excretion of lead has been used to construct compartmental pharmacokinetic models that describe the concentration of lead in various pools in the body and can be used to assess risks to individuals exposed to lead in soils and other media. Since the concentration of lead in blood is in equilibrium with the other pools of lead in the body, it is a good biomarker for exposure and can be used in the models as a predictor of risk due to exposures to lead.

4.7.1 Child Exposure to Lead

We evaluate the potential for adverse health effects in children due to lead exposure using the USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model. This is a pharmacokinetic model that takes into account multi-media exposures of young children (ages less than 6 months to 6 years). This population is most sensitive to lead's effects due in part to physiological characteristics (e.g., efficient absorption and developing nervous system/blood brain barrier) and to behavior patterns (e.g., hand-to-mouth and frequent ingestion of soils).

The model calculates blood lead levels based on children's estimated exposure to lead from various media such as food, soil, dust, and water. The output of the IEUBK model is a predicted distribution of blood lead levels in children from ages less than 6 months to 6 years of age. From this distribution, the model calculates the probability that blood lead concentrations will exceed 10 μg lead per deciliter of blood. The Center for Disease Control (CDC) established this blood lead level goal of 10 μg/dl to prevent impairment of cognitive and behavioral development (CDC 1991).

The IEUBK model does not evaluate exposures via inhalation of fugitive dust derived from soil. Lead particles larger than 0.5 μm are not well absorbed by the lungs, and this pathway typically is an insignificant contributor to the overall intake (less than 2%) when compared with ingestion. The USEPA did not include this pathway when developing the IEUBK model. Dust exposures in the model are via ingestion of indoor dust derived, at least in part, from soil.

A detailed description of the IEUBK model can be found in USEPA's documentation and guidance for its use (USEPA, 1994 a,b,c; USEPA 1998b). Appendix M includes model input parameters and output for

this assessment. In computing the blood lead levels, we use the "Single Run With Current Parameters" option in the IEUBK Model Software. We present the output of the IEUBK model in tabular form (lead intake concentrations for each medium and the blood lead values by age range). Also, the data are presented graphically as the cumulative probability distribution for exceeding a blood lead level of 10 μg/dl.

4.7.2 Adult Exposure to Lead

We use a modeling approach to evaluate adult (and adolescent) risk from lead exposure (US EPA 1996b). The model is referred to as the US EPA Technical Review Workgroup (EPA TRW) Model. It is a biokinetic model that estimates uptake of lead ingested incidentally with soil. The model is typically used to evaluate exposure for women of child-bearing age because a developing fetus is considered a sensitive receptor. Lead is efficiently transferred across the placental membranes. The lead concentration in human umbilical cord blood is 85 to 90% that of maternal blood, and lead accumulation in fetal tissues is proportional to maternal blood lead levels (Goyer, 1990). The mean concentration of lead in umbilical cord blood from a sample of over 11,000 women was 6.6 +/- 3.2 μg/dL (Bellinger et al., 1987).

The model calculates the amount of lead in the blood of women as well as the amount of lead in the blood of the fetus. A target fetal blood lead level of 10 μg/dl is used to evaluate whether there is a risk to the fetus. This target level is the same as that used to evaluate potential risks to young children. Because adults are considered less sensitive to lead exposure than children, the target level for children is also protective of adults.

The US EPA TRW methodology explicitly considers protection of the 95th percentile of the population. This means that out of an exposed population of, for example, a thousand people, 950 of the exposed people would have blood lead values less than the predicted 95th percentile value. The model often predicts blood lead concentrations greater than the 95th percentile because many of the exposure values used in the assessment represent upper percentiles of the distributions for these values in the population.

Equation A-7 from the US EPA TRW report (1996b) presents the equation for predicting blood lead levels for fetuses in women exposed to lead at the Site:

$$PbB_{fetal,0.95} = R_{fetal/maternal} \times GSD_i^{1.645} \times \left[\frac{PbS \times BKSF \times IR_S \times AF_S \times EF_S}{AT} + PbB_{adult,0} \right]$$

Where:

$PbB_{fetal,0.95}$ is the 95th percentile blood lead concentration (μg/dl) among fetuses born to women having exposures to the specified site soil lead concentrations;

$R_{fetal/maternal}$ is the ratio between the fetal blood lead concentration at birth and the maternal blood lead concentration. The factor $R_{fetal/maternal}$ is used to relate adult blood level to fetal blood. The US EPA TRW uses a value of 0.9 for $R_{fetal/maternal}$.

GSD_i is the individual geometric standard deviation in blood lead concentrations among adult women who have similar lead exposures. According to the US EPA TRW report, the GSD of the distribution of blood lead levels ranges between 1.8 for homogeneous populations to 2.1 for heterogeneous populations. We note that lower values have been reported in the literature.

Because we selected a geometric mean value of 2 μg/dl to reflect an ethnically-mixed population of women (see description of $PbB_{adult,0}$ below), we use the higher GSD value (2.1) to reflect a heterogeneous population.

PbS is the lead EPC in soil to which an individual is exposed (in μg/g).

BKSF is the quasi-steady state biokinetic slope factor relating increase in adult blood lead concentrations to average daily uptake (in μg/dl blood lead increase per μg/day of lead uptake). The value given in the EPA TRW report is 0.4.

IR_s is the ingestion rate of soil and soil-derived dust (in g/day). In this assessment, this value varies by exposure scenario.

AF_s is the absolute gastrointestinal absorption fraction for ingested lead in soil and dust. A value of 0.12 is used in the model based on an absorption factor for soluble lead of 0.20 and a relative bioavailability of 0.6 as described in the US EPA TRW report.

EF_s is the exposure frequency for contact with soils (in days of exposure during the averaging period or days per year for continuing, long term exposure).

AT is the averaging time (365 days/year for continuing long term exposures).

$PbB_{adult,0}$ is the baseline adult blood lead concentration. Statistics on blood lead concentrations for women have been derived from epidemiological studies and are presented in the US EPA TRW report. The central estimate of blood lead level is reported to range between 1.7 and 2.2 μg/dL with white women having lower levels than Mexican American and non-Hispanic black women. We propose to protect an ethnically-mixed population of women and propose to use a central estimate of blood lead (2 μg/dl).

This model provides an estimate of the upper 95th percentile fetal blood lead concentration by first calculating the adult central tendency blood lead concentrations from the adult baseline blood lead concentration ($PbB_{adult,0}$) combined with lead intake from exposure to Site soils. The upper 95th percentile of this estimated adult blood lead concentration is calculated by multiplying it by the individual geometric mean (GSD_i) raised to the power of 1.65. This latter calculation assumes that the GSD_i of the population has not been affected by Site-related exposure to lead. The risk calculations for lead using the EPA TRW model are presented in Appendix M.

4.7.3 Comparison to WHO Tolerable Daily Intake for Lead

We compare the estimated lead intakes with a TDI developed by WHO (1995). A Provisional Tolerable Weekly Intake (PTWI) of 25 μg/kg body weight is recommended by the Joint FAO/WHO Expert Committee on Food Additives and Food Contaminants (FAO/WHO, 1993). This level refers to lead from all sources and was set to protect human health, including infants and children. It is based on a model that indicates daily intakes of lead between 3 to 4 μg lead/kg body weight by infants and children are not associated with an increase in blood lead concentrations.

4.8 Risk Associated with Exposure to Petroleum Fractions (VPH/EPH)

Sites contaminated with petroleum are difficult to evaluate because the composition and distribution of complex petroleum products change following release to the environment. Individual compounds partition differently among environmental media and degrade due to processes such as photolysis and microbial action. For this reason, basing site decisions on whole product (e.g., gasoline, #2 fuel oil, jet fuel) or total petroleum hydrocarbon (TPH) data may be appropriate for fresh spills, but not for older spills that have had time to weather. Moreover, TPH composition depends on the type of petroleum contamination at a site, and TPH measurements can vary according to the analytical method used.

For these reasons, petroleum fraction-based approaches are emerging. The fraction method involves dividing petroleum mixtures into fractions and assigning representative toxicity criteria to each fraction. Use of these fractions provides several benefits. Unlike whole product or TPH data, fraction data account for the age and environmental weathering of spilled product. Fractions can be used to address any type of petroleum contamination, regardless of whether one or more petroleum products were released to the environment. Quantifying fractions also represents a practical alternative to evaluating hundreds of individual petroleum compounds. Furthermore, the toxicity data and fate and transport properties needed for assessing health risk are not available for many petroleum hydrocarbons.

The Massachusetts Department of Environmental Protection (MA DEP) was first in using petroleum fractions to characterize and evaluate potential health risk (MA DEP 1997), followed by British Columbia Environment and the TPH Criteria Working Group (the "Working Group). The Working Group divided petroleum into 13 fractions according to expected transport properties of individual compounds (Gustafson et al. 1996) and developed RfCs and RfDs for these fractions (Edwards et al. 1997). To determine the toxicity of petroleum fractions, the Working Group gathered toxicity studies for whole products, petroleum mixtures, and individual petroleum compounds. Unlike MA DEP and some other state regulatory authorities, the Working Group chose not to use the toxicity of a single reference compound to represent the toxicity of each fraction. Instead, the Working Group reviewed all available data applicable to each fraction, prioritizing mixture data. Mixture data were given higher priority in developing toxicity criteria because they account for compound interactions within the fractions. With these data, the Working Group developed reasonably conservative reference concentrations (RfCs) and reference doses (RfDs) that account for uncertainty in the underlying toxicity database (Edwards et al. 1997). RfDs and RfCs were developed following the USEPA methodology, except where otherwise noted in Edwards et al. 1997.

Site media were analyzed for six fractions using the MA DEP volatile petroleum hydrocarbon/extractable petroleum hydrocarbon (VPH/EPH) analytical method. RfDs and RfCs developed by the Working Group are used with these fraction data to estimate non-cancer hazard at the Site. These toxicity values are provided in Tables 5.1 and 5.2. In some cases, the same toxicity criterion was assigned to multiple fractions if the fractions are likely to exhibit similar toxicity.

Use of the petroleum fraction RfDs and RfCs requires several assumptions:

- Fraction toxicity will not vary significantly from the compound or mixture used to develop the toxicity criterion for the fraction. Toxicity criteria are designed to account for uncertainty in the underlying toxicity database by overestimating rather than underestimating fraction toxicity.
- Application of each toxicity criterion is appropriate whether or not the compound or mixture from which the toxicity criterion was derived is present in environmental samples. This

assumption is reasonable because the Working Group relied on toxicity data that represent the toxicity of the entire fraction rather than the material tested.

- The toxicity of a given fraction does not change with different petroleum product sources. For example, the toxicity of the $C_{>10}$ to C_{12} aliphatic fraction measured at a gasoline spill site is the same as the toxicity of the $C_{>10}$ to C_{12} aliphatic fraction measured at a #2 fuel oil spill site. This assumption is based on the fact that petroleum products represent different distillation cuts from crude oil, although crude oil composition is variable and products contain different additives and blending agents.

4.9 Evaluation of Risk for Some Special Case Metals

Chromium data was assumed to all be in the form of chromium VI. This is likely to be a conservative assumption. The IRIS file for chromium VI lists different RfCs for chromium in aerosols vs. chromium as particulates. Therefore, in this assessment, we used the RfC for chromium in aerosols to estimate risk from the drinking water pathways for the future resident. We used the RfC for chromium as particulates to estimate risk from soil and sediment pathways.

The IRIS file for cadmium lists different RfDs for cadmium ingested in food and cadmium ingested in water. Therefore, in this assessment, we used the oral RfD for cadmium in food to estimate risk from the soil pathways and the oral RfD for cadmium in water to estimate risk from the drinking water pathways.

The IRIS file for manganese recommends that a modifying factor of 1 be applied to the oral RfD when assessing exposure to manganese from food (fish ingestion in this assessment). USEPA Region 1 (1999) explains that the oral RfD (1.4×10^{-1}) represents the allowable level for total oral intake (i.e., intake from multiple site media). Therefore, when estimating risk from oral intake of soil and groundwater, USEPA Region 1 suggests subtracting the dietary contribution from the total allowable intake. Therefore, we used the non-dietary reference dose of 7×10^{-2} mg/kg/day when calculating risk from soil exposures. For exposures to groundwater, a modification factor of 3 was applied to the non-dietary reference dose, resulting in a groundwater reference dose of 2.4×10^{-2}.

5.0 RISK CHARACTERIZATION

The purpose of the risk characterization is to estimate potential risks associated with Site contaminants for each exposure scenario. The results of the dose-response assessment are combined with the results of the exposure assessment to derive quantitative estimates of risk and hazard for carcinogenic and noncarcinogenic COPCs, respectively.

The risk characterization compares estimated Site-specific risk levels to target risk levels. The EPA target cancer risk range is 10^{-4} to 10^{-6}, while the NHDES target risk level is 1×10^{-5}. USEPA and NHDES share the same target non-cancer hazard index of 1.

5.1 Non-cancer Hazard Evaluation

We evaluate the potential for non-cancer health effects by calculating hazard quotients (HQs) and hazard indices (HIs). The HQ is the quotient of the average daily dose (ADD) for a given exposure pathway to the chemical- and route-specific (oral, dermal, or inhalation) reference dose (RfD).

$$HQ = ADDi/RfDi$$

Where:
ADDi = Average daily dose of contaminant i; estimated daily intake averaged over the exposure period (mg/kg-day)
RfD = Reference dose (mg/kg-day)

HQs are summed across COPCs to estimate a pathway-specific HI. These pathway-specific HIs are summed to account for a single human receptor's multiple pathway exposure.

RAGS Part D Table 7s (Appendix N) summarize the results of the non-cancer hazard evaluation. These tables show the inputs used to calculate the ADD, the toxicity information used, and the non-cancer hazard for each unique combination of scenario timeframe, medium, exposure medium, exposure point, receptor population, and receptor age. There are two totals at the bottom of each Table 7. One is the sum of the hazard across all COPCs and direct exposure pathways (i.e. ingestion and dermal contact). The other is the sum of the hazard across all COPCs and all exposure pathways (the direct exposure pathways and modeled exposure pathways (i.e., inhalation exposure pathways; see Appendix I). Total hazard indices do not include the garden pathway.

Non-cancer hazard estimates are not shown for COPCs that do not have any available toxicity information (e.g., alkylbenzenes and carcinogenic PAHs in soil, MTBE in groundwater). See section 4.4 for a discussion of COPCs without toxicity criteria. Also, non-cancer hazard from lead is not included in the RAGS Part D Table 7s. See section 4.7 for an explanation of how we evaluated risk from lead exposure for different receptors at the Site.

5.2 Cancer Risk Evaluation

Cancer risk is calculated by multiplying the chemical-specific lifetime average daily dose (LADD) through a particular exposure route by the exposure-route-specific (oral, inhalation or dermal) cancer slope factor (CSF), as shown in the following equation.

$$\text{Excess Lifetime Cancer Risk} = \text{LADDi} * \text{CSFi}$$

Where:

LADDi = Lifetime average daily dose of contaminant i; intake averaged over a 70-year lifetime in mg chemical/kg body weight per day (mg/kg-day)
CSFi = Chemical- and route-specific cancer slope factor of contaminant i $(\text{mg/kg-day})^{-1}$

RAGS Part D Table 8s (See Appendix O) include cancer risk estimates (USEPA, 1997a). These tables summarize the inputs used to calculate the LADD, the toxicity values, and the cancer risk for each unique combination of scenario timeframe, exposure medium, exposure point, receptor population, and receptor age. There are two totals at the bottom of each Table 8. One is the sum of the risk across all COPCs and direct exposure pathways (i.e. ingestion and dermal contact). The other is the sum of the risk from all COPCs for all exposure pathways, including the direct exposure pathways and modeled exposure pathways (i.e., inhalation exposure pathways; see Appendix I). Total risk estimates do not include the garden pathway.

RAGS Part D Table 8s (Appendix O) show cancer risk estimates only for COPCs that are classified as A, B, or C carcinogens. Some COPCs are classified as carcinogens, but only have inhalation toxicity data. The risk from each of these compounds via the inhalation pathway is not shown on the RAGS Part D tables, but this risk is included in the sum of total risk across all exposure pathways.

5.3 Risk Summary

This section presents a summary of risk estimates for each exposure scenario considered in this risk assessment. We assume that risk is additive across all exposure routes for all COPCs. Hazard and risk calculated from modeled EPCs are presented separately from risk estimated using measured EPCs in the summary tables below.

RAGS Part D Table 9s and 10s (in Appendices P and Q, respectively) provide more detailed information about carcinogenic risks and non-cancer hazards for each receptor. Table 9s show the risk from all COPCs for which cancer risk or non-cancer hazard were calculated. The COPCs for which the cancer risk exceeds 1×10^{-6} and the non-cancer hazard exceeds the target HI of 1 are summarized in RAGS Part D Table 10s (EPA, 1998b).

5.3.1 Future Resident

The future resident represents the most sensitive receptor considered in the risk assessment. This is due to the increased number of pathways by which this receptor is exposed as well as an increased exposure

frequency and duration, compared to other receptors. A hypothetical future resident is exposed to soil via direct contact and inhalation of fugitive dust and vapors, and to contaminants in tap water via direct contact and inhalation of vapors. A qualitative approach is used to estimate the hazard and risk from groundwater vapor inhalation. Following EPA Guidance, we assume that the groundwater hazard and risk from VOCs via the ingestion pathway is equal to the groundwater inhalation hazard and risk (USEPA Region 1 Risk Update, 1995).

We calculated two soil EPCs for the future resident; one for 0 to 10 foot soil and one for 0 to 1 foot soil. Following USEPA guidance, we assumed vertical mixing of soil in the future and calculated an EPC for soil from the surface down to a depth of 10 feet, which EPA considers the depth of excavation for building a foundation. While there are areas of subsurface contamination at the Site, much of the contamination is at the surface. Therefore, we also calculated a 0-1 ft soil EPC. In general, the surface soil is more highly contaminated than subsurface soil and people are more likely to be exposed to what is in the surface soil. The 0 to 1 foot surface soil EPC is also representative of a future resident's exposure assuming little soil mixing. Soil samples on Parcel 1 and at or near the soil piles on Parcel 2 were included in the soil EPC calculations. The future groundwater EPC includes groundwater data from both parcels, excluding the background wells and off-Site unimpacted well locations.

A summary of risk for the future resident is shown in Table 5. Risk is calculated for both the RME and CT cases. The cancer risk and non-cancer hazard to both child and adult residents for the RME and CT cases exceed EPA target risk levels. Cancer risk from the RME case is 2 orders of magnitude higher and the hazard index is 5 - 10 times higher than the CT case. It is possible that someone could spend the first thirty years of their life living at the Site; therefore, the child and adult cancer risk estimates are summed in the last portion of Table 5.

We evaluate risk to the future resident from exposure to groundwater and to soil at depths of 0-1 foot and 0-10 feet. The total hazard index for both the child and adult resident is higher for exposure to 0-1 ft soil than for exposure to 0-10 ft soil. This result is due to the fact that PCBs are the major contributor to non-cancer hazard and the EPC for PCBs in surface soil is larger than in 0-10 ft soil. The total cancer risk for both the child and adult resident is the same in 0-1 and 0-10 ft soil (based on one significant figure).

A detailed summary of risk and hazard estimates for all COPCs for the future resident are presented Tables 9.1 through 9.4 Appendix P. Tables 10.1 through 10.4 in Appendix Q show only the COPCs with risk or hazard that exceeds target risk levels (i.e. risk drivers). Table 6 lists the risk drivers for the RME future child resident.

Non-Cancer Hazard from Future Child Resident Exposure to Lead

We evaluate the potential for adverse health effects in children due to lead exposure using the USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model. The model calculates blood lead levels based on children's estimated exposure to lead from various media such as food, soil, dust, and water. We entered a Site-specific soil lead concentration into the model. We ran the model twice, once for each soil EPC. The lead EPC in 0-1 ft soil is 895 mg/kg and the lead EPC in 0-10 ft soil is 491 mg/kg. The 95% UCL on the mean is the EPC for both the RME and CT cases.

Table 5. Summary of Non-cancer Hazard and Cancer Risk Estimates for a Future Resident

Scenario	Exposure Pathway	Chronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	*CT*	*RME*	*CT*
Future Adult Resident Exposed to soil (0 to 10 feet) via direct contact and inhalation of fugitive dust and vapors, and to tap water via direct contact and inhalation of vapors.	**Soil Oral**	4	2	1.E-04	1.E-05
	Soil Dermal	2	0.3	5.E-05	2.E-06
	Soil Inhalation	0.02	0.003	4.E-07	2.E-09
	Groundwater Oral	30	4	2.E-02	1.E-04
	Groundwater Dermal	20	0.7	1.E-03	4.E-06
	Groundwater Inhalation	20	0.6	2.E-02	9.E-05
	Total	80	7	4.E-02	2.E-04
	% Risk from soil	8%	28%	0.4%	5%
	% Risk from groundwater	92%	72%	99.6%	95%
Future Adult Resident Exposed to soil (0 to 1 foot) via direct contact and inhalation of fugitive dust and vapors, and to tap water via direct contact and inhalation of vapors.	**Soil Oral**	10	6	3.E-04	2.E-05
	Soil Dermal	7	1	2.E-04	3.E-06
	Soil Inhalation	0.08	0.002	5.E-07	3.E-09
	Groundwater Oral	30	4	2.E-02	1.E-04
	Groundwater Dermal	20	0.7	1.E-03	4.E-06
	Groundwater Inhalation	20	0.6	2.E-02	9.E-05
	Total	90	12	4.E-02	2.E-04
	% Risk from soil	22%	60%	1%	9%
	% Risk from groundwater	78%	40%	99%	91%
Future Child Resident Exposed to soil (0 to 10 feet) via direct contact and inhalation of fugitive dust and vapors, and to tap water via direct contact and inhalation of vapors.	**Soil Oral**	30	20	2.E-04	3.E-05
	Soil Dermal	20	4	9.E-05	6.E-06
	Soil Inhalation	0.04	0.007	3.E-07	1.E-09
	Groundwater Oral	110	10	2.E-02	1.E-04
	Groundwater Dermal	30	1	6.E-04	2.E-06
	Groundwater Inhalation	70	2	2.E-02	7.E-05
	Total	260	30	3.E-02	2.E-04
	% Risk from soil	19%	59%	1%	16%
	% Risk from groundwater	81%	41%	99%	84%
	Lead: children >10µg/dL blood	36%			
Future Child Resident Exposed to soil (0 to 1 foot) via direct contact and inhalation of fugitive dust and vapors, and to tap water via direct contact and inhalation of vapors.	**Soil Oral**	120	60	7.E-04	5.E-05
	Soil Dermal	50	10	3.E-04	1.E-05
	Soil Inhalation	0.2	0.005	3.E-07	2.E-09
	Groundwater Oral	120	10	2.E-02	1.E-04
	Groundwater Dermal	30	1	6.E-04	2.E-06
	Groundwater Inhalation	70	2	2.E-02	7.E-05
	Total	380	80	3.E-02	2.E-04
	% Risk from soil	44%	84%	3%	26%
	% Risk from groundwater	56%	16%	97%	74%
	Lead: children >10µg/dL blood	70%			
Future Child Resident and Future Adult Resident Combined Exposed to soil (0 to 10 feet)				7E-02	4E-04
Exposed to soil (0 to 1 foot)				7E-02	4E-04

Table 6. "Risk Drivers" for the RME Future Child Resident

Medium	Cancer Risk Drivers [1]	Percent of Total Cancer Risk [2]	Non-cancer Risk Drivers [1]	Percent of Total Non-cancer Hazard [2]
Soil	Total PCBs and Dioxin-like PCB congeners	2% (0-10'); 6% (0-1')	Total PCBs	21% (0-10'); 50% (0-1')
	Bis(2-ethylhexyl)phthalate	<1%	C_{11}-C_{22} aromatic fraction	1%
	Benz(a)anthracene	<1%	Chromium VI	2% (0-10'); 1% (0-1')
	Benzo(a)pyrene	<1%	Lead	NI
	Dibenz(a,h)anthracene	<1%	Mercury	1% (0-10')
	Arsenic	<1%	Nickel	<1%
Groundwater	Benzene	<1%	Benzene	18% (0-10'); 11% (0-1')
	1,2-Dichloroethane	<1%	1,1-Dichloroethane	<1%
	1,1-Dichloroethene	<1%	cis-1,2-Dichloroethene	13% (0-10'); 8% (0-1')
	Methylene chloride	<1%	Ethylbenzene	3% (0-10'); 2% (0-1')
	1,1,2,2-Tetrachloroethane	<1%	Methylene chloride	2% (0-10'); 1% (0-1')
	Tetrachloroethene	<1%	Toluene	<1%
	Trichloroethene	1%	1,1,1-Trichloroethane	2% (0-10'); 1% (0-1')
	Vinyl chloride	89% (0-1'); 93% (0-10')	Vinyl chloride	7% (0-10'); 5% (0-1')
	Aldrin	<1%	C9-C10 aromatic fraction	1%
	alpha-BHC	<1%	C11-C22 aromatic fraction	2% (0-10'); 1% (0-1')
	gamma-BHC	<1%	Naphthalene	7% (0-10'); <1% (0-1')
	Dieldrin	<1%	Antimony	1% (0-10'); 1% (0-1')
	Heptachlor	<1%	Arsenic	5% (0-10'); 3% (0-1')
	Heptachlor epoxide	<1%	Cadmium	1%
	Arsenic	2%	Chromium VI	2% (0-10'); 1% (0-1')
			Manganese	14% (0-10'); 9% (0-1')

NI = Not included in calculation of percent risk

(1) Risk driver compounds have a hazard index that exceeds one or a cancer risk that exceeds 1E-06.

(2) The table above shows the percent risk and percent hazard via the direct exposure pathways (ingestion and dermal contact with soil and groundwater) for the future child resident RME scenario.

Appendix M includes model input parameters and output for this assessment. In computing the blood lead levels, we use the "Single Run With Current Parameters" option in the IEUBK Model Software. We present the output of the IEUBK model in tabular form (lead intake concentrations for each medium and the blood lead values by age range). Also, the data are presented graphically as the cumulative probability distribution for exceeding a blood lead level of 10 μg/dl. The Center for Disease Control (CDC) established this blood lead level goal of 10 μg/dl to prevent impairment of cognitive and behavioral development (CDC 1991).

For the future child resident exposed to the lead EPC in 0-1 ft soil (the 95% UCL on the mean), 70% of children are predicted to have blood lead levels greater than 10 μg lead per deciliter of blood. For the future child resident exposed to the lead EPC in 0-10 ft soil (again, the 95% UCL on the mean), 36% of children are predicted to have blood lead levels greater than 10 μg lead per deciliter of blood.

WHO Lead Weekly Tolerable Intake Comparison

The future child RME daily intake rate of 5.3 μg/kg/d for 0-1 ft soil would lead to an exceedance of the PTWI for lead of 25 μg/kg/d.

Non-Cancer Hazard from Future Adult Resident Exposure to Lead

USEPA Region 1 (1996) advises that only a child's exposure to lead need be evaluated for future residential exposure scenarios. Therefore, we did not quantify risk from future adult residential exposure to lead.

Nitrate

The maximum nitrate-nitrogen concentration in groundwater samples used to estimate non-cancer hazard for a future resident is 6 mg/L. The resulting hazard quotient was less than one.

However, one groundwater sample not used to estimate the EPC for a future resident contains 14 mg nitrate-nitrogen/L (sample #SH-24I), which exceeds the NH AGQS of 10 mg/L. This sample did not appear to be impacted by Site-related contamination, which is why it was excluded from the EPC calculation. The RI (pg. 106, para. 2) indicates that this contamination may have resulted from surface water runoff from a nearby roadway. The concentration falls in the LOAEL range of 11-20 mg nitrate-nitrogen/L (USEPA 1999a) identified for nitrate-nitrogen and may be of concern if people use groundwater with nitrate concentrations in this range as a drinking water source in the future.

WHO Dioxin TEQ Tolerable Daily Intake Comparison

Predicted daily intakes for the future resident greatly exceed the TDI given elevated concentrations of dioxin-like PCB congeners.

Compounds that Exceed Secondary Drinking Water Standards

Chloride and iron are not COPCs in groundwater, therefore they are not evaluated in the risk assessment. However, a future resident may be exposed to concentrations of chloride and iron in groundwater, which may cause cosmetic effects (e.g, skin or tooth discoloration) or aesthetic effects (i.e., taste, odor, or color). The chloride concentration in samples SH-24S, SH-22S, and SH-14D (390, 360 and 290 mg/L, respectively) exceeds the EPA recommended National Secondary Drinking Water Standard for chloride of 250 mg/L. Iron was analyzed in 26 groundwater samples. The concentration of iron in 22 of these samples (ranging from 0.34 to 110 mg/L) exceeds the EPA National Secondary Drinking Water Standard for iron of 0.3 mg/L. The National Secondary Drinking Water Standards are non-enforceable guidelines, however they can be used to identify concentrations in groundwater, which may cause cosmetic or aesthetic effects. (EPA Office of Water website, 3/99.)

Manganese concentrations in groundwater also exceed the EPA recommended Secondary Drinking Water Standard for manganese (0.05 mg/L) in 17 of 20 samples. However, manganese is a COPC in groundwater for the future resident, and the risk-based preliminary remediation goal (PRG) of 0.02 mg/L (MCA, 2000) is less than the Secondary Drinking Water Standard for manganese. Therefore, a future resident will be protected against organoleptic effects if this PRG is attained.

5.3.2 Current Resident

We evaluate the potential risk to a current resident exposed to groundwater from two wells with point-of-entry treatment systems that draw from Site groundwater. These wells have treatment systems because they have been impacted by contaminated groundwater from the Site. Current residents are drinking treated groundwater; however, we used pre-treatment system samples to illustrate the risk residents might incur in the absence of these treatment systems. We estimated risk using validated groundwater data collected in 1997.

A current resident may be exposed to contaminants in untreated tap water via ingestion, dermal contact, and vapor inhalation. A qualitative approach is used to estimate the hazard and risk from groundwater vapor inhalation. Following EPA Guidance, we assume that the hazard and risk from VOCs via the ingestion pathway is equal to the vapor inhalation hazard and risk (USEPA Region 1 Risk Update, 1995).

Risk is calculated for both the RME and CT cases. RME risk estimates are based on a combination of maximum COPC concentrations among all wells. The cancer risk to both child and adult residents for the RME and CT cases are within the EPA target risk range of 10^{-4} to 10^{-6}. The cancer risk for the CT case is 9 to 10 times lower than the RME case. Non-cancer HIs do not exceed the target HI of one for the child or the adult resident. A summary of risk for the current resident is shown in Table 7.

A detailed summary of risk and hazard estimates for all COPCs for the current resident are presented in Tables 9.5 and 9.6 in Appendix P. Tables 10.5 and 10.6 in Appendix Q show only the COPCs with risk or hazard that exceeds target risk levels (i.e. risk drivers). Vinyl chloride is the primary contributor to cancer risk for the child and adult current resident (96%) and is the only risk driver COPC.

Table 7. Summary of Non-cancer Hazard and Cancer Risk Estimates for the Current Resident

Scenario	Exposure Pathway	Chronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	***CT***	***RME***	***CT***
Current Adult Resident Exposed to contaminants in pretreatment tap water from two wells with treatment systems that draw from Site groundwater via ingestion, dermal contact and vapor inhalation.	**Groundwater Oral**	0.1	0.05	5.E-05	6.E-06
	Groundwater Dermal	0.02	0.005	4.E-06	2.E-07
	Groundwater Inhalation	0.1	0.05	5.E-05	6.E-06
	Total	0.2	0.1	1.E-04	1.E-05
	% Risk from groundwater	100%	100%	100%	100%
Current Child Resident Exposed to contaminants in pretreatment tap water from two wells with treatment systems that draw from Site groundwater via ingestion, dermal contact and vapor inhalation.	**Groundwater Oral**	0.4	0.2	4.E-05	5.E-06
	Groundwater Dermal	0.04	0.009	2.E-06	1.E-07
	Groundwater Inhalation	0.4	0.2	4.E-05	5.E-06
	Total	0.8	0.3	9.E-05	1.E-05
	% Risk from groundwater	100%	100%	100%	100%

While it is true that hazard index estimates are less than one and cancer risk estimates fall within the USEPA's target risk range of 10^{-4} to 10^{-6}, the cancer risk estimate of 10^{-4} exceeds NHDES's target cancer risk level of 1E-5. Furthermore, we used only validated data available for the wells with point-of-entry treatment systems. When examining VOC concentration data over time, a generally increasing trend is observed, most notably for 1,2-DCE in wells with treatment systems. Concentrations of groundwater

COCs measured in some of the wells sampled during July 2000 are about two times higher than concentration in 1997. Also, it should be noted that some of these concentrations exceed NH AGQSs. Given these trends, higher risk would be estimated using the most recent (unvalidated) concentration data rather than the 1997 concentration data.

This trend of increasing VOC concentrations over time occurs in untreated residential wells in the area, as well. Therefore, sentinel wells would be useful for predicting if Site contamination might impact additional residential wells.

PCBs in the Yard Soil of Adjacent Residences

Soil samples were collected from nine residential backyards believed to be most likely to be contaminated by Site-related PCBs (ATSDR, 1996b). PCBs were not detected in 26 of 28 samples collected, with a detection limit of 22 µg/kg. Two samples with detectable levels of PCBs were collected from the same residence (170 µg/kg Aroclor 1260 and 140 µg/kg Aroclor 1254).

In 1998, four soil samples were collected on Parcel 1 near Kelley Road (S-200, S-201, S-202, and S-203) and analyzed for PCBs to determine if Site contamination might impact residential properties on the other side of the road. PCBs were very weathered in these samples and did not match the characteristic pattern of any Aroclor mixture. Therefore, the laboratory noted this result and reported the samples as nondetect for the six target Aroclors. However, the validator estimated J-qualified PCB concentrations in these samples of 150, 240, 840, and 1080 µg/kg quantified as Aroclor 1242, the Aroclor mixture most prevalent at the Site. All detected PCB concentrations are less than or approximately equal to the NHDES S-1 standard of 1,000 µg/kg.

5.3.3 Future Construction Worker

A future construction worker may be exposed via direct contact with soil and groundwater, inhalation of dust and vapors from soil, and inhalation of vapors from groundwater. Soil samples between 0 and 10 feet on Parcel 1 and on Parcel 2 at or near the soil piles were used to develop the soil EPC for the construction worker. Groundwater data collected at less than 15 feet below ground surface on Parcels 1 and 2, excluding wells in which all COPCs were never detected, were used to calculate the groundwater EPC for the construction worker.

Risk is calculated for both the RME and CT cases. The RME cancer risk to the construction worker exceeds the upper end of the EPA target risk range of 10^{-4} to 10^{-6}. The CT cancer risk is within this target risk range. Both RME and CT hazard exceed the EPA target HI of one, but the hazard index for the CT case is 4 times lower than the RME case. A summary of risk for the future construction worker is shown in Table 8.

A detailed summary of risk and hazard estimates for all COPCs for the current resident are presented in Tables 9.5 and 9.6 in Appendix P. Tables 10.5 and 10.6 in Appendix Q show only the COPCs with risk or hazard that exceeds target risk levels (i.e. risk drivers). Vinyl chloride is the primary contributor to cancer risk for the child and adult current resident (96%) and is the only risk driver COPC.

Table 8. Summary of Non-cancer Hazard and Cancer Risk Estimates for the Future Construction Worker

Scenario	Exposure Pathway	Subchronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	***CT***	***RME***	***CT***
Future Construction Worker Exposed to soil (0 to 10 feet) via direct contact and inhalation of fugitive dust and vapors. Exposed to groundwater in an excavation area via ingestion and dermal contact.	**Soil Oral**	20	9	2.E-05	8.E-06
	Soil Dermal	4	2	4.E-06	2.E-06
	Soil Inhalation	0.1	0.02	1.E-07	2.E-09
	Groundwater Oral	0.2	0.04	8.E-06	5.E-07
	Groundwater Dermal	30	2	2.E-04	7.E-06
	Total	50	10	2.E-04	2.E-05
	% Risk from soil	37%	82%	12%	56%
	% Risk from groundwater	63%	18%	88%	44%

Table 9. "Risk Drivers" for the Future Construction Worker

Medium	Cancer Risk Drivers (1)	Percent of Total Cancer Risk (2)	Non-cancer Risk Drivers (1)	Percent of Total Non-cancer Hazard (2)
Soil	Total PCBs and Dioxin-like PCB congeners	10%	Total PCBs	33%
Groundwater	Benzene	4%	Benzene	39%
	Tetrachloroethene	3%	Ethylbenzene	15%
	Vinyl chloride	80%	Manganese	5%

(1) Risk driver compounds have a HI that exceeds one or a cancer risk that exceeds 1E-06.
(2) The table above shows the percent risk and percent hazard via the direct exposure pathways (ingestion and dermal contact with soil and groundwater) for the future construction worker RME scenario.

Non-Cancer Hazard from Future Construction Worker Exposure to Lead

USEPA Region 1 (1996) recommends evaluation of lead exposure for construction workers using the USEPA's Technical Review Workgroup (EPA TRW) Model (US EPA 1996b). The model calculates blood lead levels in women of child-bearing age based uptake of lead ingested incidentally with soil. It also calculates the 95th percentile blood lead concentration among fetuses born to women exposed to site soil lead concentrations. A target fetal blood lead level of 10 μg/dl is used to evaluate whether there is a risk to the fetus. We entered a Site-specific soil lead concentration into the model.

Given uncertainty about the soil ingestion rate for a construction worker, we ran the model using two ingestion rates: USEPA's conservative screening value of 480 mg/day and 100 mg/day, which is suggested for "contact intensive scenarios" in USEPA's "Frequently Asked Questions (FAQs) on the Adult Lead Model" (USEPA 1999a). Using model inputs listed in Section 4.7.2 above, a 100 mg/day ingestion rate, lead EPCs for 0-1 ft soils and 0-10 ft soils, and the CT exposure frequency of 90 days, the 95th percentile fetal blood lead concentration does not exceed 10 μg lead per deciliter of blood. Using the 480 mg/day ingestion rate, lead EPCs for 0-1 ft soils and 0-10 ft soils, and the CT exposure frequency, the 95th percentile fetal blood lead concentration does exceed 10 μg lead per deciliter of blood.

5.3.4 Current Trespasser

The current trespasser is an adolescent who may be exposed to contaminants in surface soil (0-1 ft depth), on Parcel 1 and on Parcel 2 adjacent to the soil piles, via ingestion, dermal contact, and inhalation of fugitive dust and vapors.

Risk is calculated for both the RME and CT cases. The RME cancer risk to the trespasser exceeds the upper end of the EPA target risk range of 10^{-4} to 10^{-6}. The CT cancer risk is within this target risk range. Both the RME and CT non-cancer hazard to the trespasser exceed the EPA target hazard index of one. A summary of risk for the current trespasser is shown in Table 10.

A detailed summary of risk and hazard estimates for all COPCs for the current trespasser are presented in Table 9.9 in Appendix P. Tables 10.9 in Appendix Q shows only the COPCs with risk or hazard that exceeds target risk levels (i.e. risk drivers). The risk drivers in surface soil for the current trespasser exposure scenario are shown in Table 11.

Table 10. Summary of Non-cancer Hazard and Cancer Risk Estimates for the Current Trespasser

Scenario	Exposure Pathway	Chronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	***CT***	***RME***	***CT***
Current Trespasser	**Soil Oral**	10	3	2.E-04	1.E-05
Exposed to soil (0 to 1 foot) via direct contact and inhalation of fugitive dust and vapors.	**Soil Dermal**	5	0.3	6.E-05	1.E-06
	Soil Inhalation	0.02	0.001	6.E-08	2.E-09
	Total	20	3	2.E-04	1.E-05
	% Risk from soil	100%	100%	100%	100%

Table 11. "Risk Drivers" for the Current Trespasser

Medium	Cancer Risk Drivers (1)	Percent of Total Cancer Risk (2)	Non-cancer Risk Drivers (1)	Percent of Total Non-cancer Hazard (2)
Soil	Total PCBs and Dioxin-like PCB congeners	97%	Total PCBs	95%
	Benzo(a)pyrene	1%		
	2,3,4,7,8-PeCDF	<1%		
	Arsenic	<1%		

(1) Risk driver compounds have a HI that exceeds one or a cancer risk that exceeds 1E-06.
(2) The table above shows the percent risk and percent hazard via the direct exposure pathways (ingestion and dermal contact with soil) for the current trespasser RME scenario.

Non-Cancer Hazard from Current Trespasser Exposure to Lead

We evaluate the potential for adverse health effects in adolescents of childbearing age due to lead exposure using the USEPA's Technical Review Workgroup (EPA TRW) Model (US EPA 1996b). The model calculates blood lead levels in women of child-bearing age based uptake of lead ingested incidentally with soil. It also calculates the 95th percentile blood lead concentration among fetuses born to women exposed to site soil lead concentrations. A target fetal blood lead level of 10 μg/dl is used to evaluate whether there is a risk to the fetus. We entered a Site-specific soil lead concentration into the model. We ran the twice for the 0-1 ft soil EPC using the RME (100 mg/day) and CT (50 mg/day) soil ingestion rates for a trespasser. The lead EPC in 0-1 ft soil is 895 mg/kg. This EPC is the 95% UCL on the mean and therefore, is the EPC for both the RME and CT cases.

The BKSF and baseline blood lead concentration are sources of uncertainty in applying this model to adolescents. Empirical data suggest that BKSFs appear to be similar for young children and adults; therefore, it is reasonable to apply the adult BKSF. Also, children 12-18 years of age reportedly have low baseline blood lead concentrations (Brody et al. 1994), which may be due to a growth spurt causing a shift of lead from blood to bone. Therefore, we used baseline blood lead concentration recommended by the TRW (USEPA 1996b).

Using model inputs listed in Section 4.7.2 above for the current trespasser exposed to the lead EPC in 0-1 ft soils, the 95th percentile fetal blood lead concentration does not exceed the target fetal blood lead concentration of 10 μg/dL for CT and RME exposure conditions.

5.3.5 Current/Future Recreational Person

The current or future recreational person is a child wading or playing in Kelley Brook. This receptor may be exposed via ingestion and dermal contact with sediment and surface water. All surface water samples in Kelley Brook (except for SW-1, SW-2 and SW-13) were used to calculate surface water EPCs. We chose sediment samples in and immediately downstream of the area where free product historically discharged to Kelley Brook (from OS-5 to OS-10) to calculate sediment EPCs.

Risk is calculated for both the RME and CT cases. The RME cancer risk to the recreational person exceeds the upper end of the EPA target risk range of 10^{-4} to 10^{-6}. The CT cancer risk is within this target risk range. Only the RME hazard index exceeds the EPA target HI of one. A summary of risk for recreation is shown in Table 12.

A detailed summary of risk and hazard estimates for all COPCs for the recreational person is presented in Table 9.8 in Appendix P. Table 10.8 in Appendix Q shows only the COPCs with risk or hazard that exceeds target risk levels (i.e. risk drivers). The risk drivers for the recreational person are presented in Table 13. Non-cancer hazard for the recreational person is primarily due to sediment exposure. Conversely, cancer risk for the recreational person is primarily due to surface water exposure.

Table 12. Summary of Non-cancer Hazard and Cancer Risk Estimates for Current/Future Recreation

Scenario	Exposure Pathway	Chronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	***CT***	***RME***	***CT***
Current/Future Recreational Person	**Surface Water Oral**	0.03	0.005	1.E-06	2.E-07
Exposed via ingestion and dermal contact with sediment and surface water.	**Surface Water Dermal**	0.2	0.04	2.E-04	1.E-05
	Sediment Oral	0.4	0.04	1.E-05	2.E-06
	Sediment Dermal	3	0.5	3.E-05	7.E-06
	Total	3	0.6	2.E-04	2.E-05
	% Risk from surface water	6%	8%	83%	55%
	% Risk from sediment	94%	92%	17%	45%

Table 13. "Risk Drivers" for Current/Future Recreation

Medium	Cancer Risk Drivers [1]	Percent of Total Cancer Risk [2]	Non-cancer Risk Drivers [1]	Percent of Total Non-cancer Hazard [2]
Surface water	Vinyl chloride	1%		
	Benz(a)anthracene	3%		
	Benzo(a)pyrene	50%		
	Benzo(b)fluoranthene	5%		
	Dibenz(a,h)anthracene	16%		
	Indeno(1,2,3-cd)pyrene	8%		
Sediment	Total PCBs and Dioxin-like PCB congeners	4%	Manganese	38%
	Arsenic	12%		

(1) Risk driver compounds have a HI that exceeds one or a cancer risk that exceeds 1E-06.
(2) The table above shows the percent risk and percent hazard via the direct exposure pathways (ingestion and dermal contact with sediment and surface water) for the recreational person RME scenario.

5.3.6 Current/Future Fisherperson

A current or future fisherperson is an adult who may be exposed via ingestion and dermal contact with sediment and surface water and via ingestion of contaminated food (fish) in Kelley Brook. All surface water samples in Kelley Brook (except for SW-1, SW-2 and SW-13) were used to calculate surface water EPCs. We chose sediment samples in and immediately downstream of the area where free product historically discharged to Kelley Brook (from OS-5 to OS-10) to calculate sediment EPCs. Brook trout data were used to calculate fish tissue EPCs.

Risk is calculated for both the RME and CT cases. The RME cancer risk to the fisherperson exceeds the upper end of the EPA target risk range of 10^{-4} to 10^{-6}. The CT cancer risk is within this target risk range. Only the RME hazard index exceeds the EPA target HI of one. A summary of risk for the fisherperson is shown in Table 14.

Table 14. Summary of Non-cancer Hazard and Cancer Risk Estimates for the Fisherperson

Scenario	Exposure Pathway	Chronic Total Hazard Index		Cancer Total Risk Estimates	
		RME	***CT***	***RME***	***CT***
Current/Future Fisherperson	**Surface Water Oral**	0.009	0.0007	8.E-07	3.E-08
Exposed via ingestion and dermal contact with sediment and surface water and via ingestion of contaminated food (fish).	**Surface Water Dermal**	0.05	0.001	1.E-04	9.E-07
	Sediment Oral	0.2	0.01	9.E-06	4.E-07
	Sediment Dermal	0. 6	0.08	1.E-05	8.E-07
	Food Oral	4	0.3	1.E-04	2.E-06
	Total	4	0.4	2.E-04	5.E-06
	% Risk from surface water	1%	<1%	47%	21%
	% Risk from sediment	18%	26%	8%	26%
	% Risk from food	81%	74%	45%	53%

A detailed summary of risk and hazard estimates for all COPCs for the fisherperson is presented in Table 9.7 in Appendix P. Table 10.7 in Appendix Q shows only the COPCs with risk or hazard that exceeds

target risk levels (i.e. risk drivers). The risk drivers for the current/future fisherperson scenario are shown in Table 15.

Table 15. "Risk Drivers" for the Fisherperson

Medium	Cancer Risk Drivers [1]	Percent of Total Cancer Risk [2]	Non-cancer Risk Drivers [1]	Percent of Total Non-cancer Hazard [2]
Surface water	Benz(a)anthracene	2%		
	Benzo(a)pyrene	28%		
	Benzo(b)fluoranthene	3%		
	Dibenz(a,h)anthracene	9%		
	Indeno(1,2,3-cd)pyrene	4%		
Sediment	Total PCBs	1%		
	Arsenic	6%		
Food	Total PCBs and Dioxin-like PCB congeners	31%	Total PCBs	54%
	Arsenic	13%		

(1) Risk driver compounds have a HI that exceeds one or a cancer risk that exceeds 1E-06.
(2) The table above shows the percent risk and percent hazard via the direct exposure pathways (ingestion and dermal contact with sediment and surface water and ingestion of fish) for the fisherperson RME scenario.

NHDHHS recommends limiting consumption of brook trout from Kelley Brook due to PCB and mercury concentrations (ATSDR 1998c). Mercury is also the subject of a statewide NHDHHS freshwater fish consumption advisory. No FDA action levels are exceeded in the edible portion of brook trout ("FDA Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed" (http://vm.cfsan.fda.gov/~lrd/fdaact.html)).

Lead was detected in 5 of 22 brook trout samples and 6 of 17 red fin pickerel samples. Whole body concentrations ranged from 0.14 to 6.2 mg/kg, fresh weight. There are no RBCs for lead in fish. The maximum concentration was detected in a fish collected from the reach of Kelley Brook adjacent to the Site (i.e., KB-3). Furthermore, we did not use the USEPA TRW model to estimate risk to fetuses from adult consumption of fish contaminated with lead given the uncertainty associated with using the model for this purpose. However, lead detected in fish may be Site-related and would likely be reduced with remediation of sediments in the oil breakout area where the highest lead concentration was measured.

One of the 23 brook trout caught in Kelley Brook was a hatchery fish. Concentrations of metals and total PCBs in this fish were not higher than the average concentration in all other brook trout. Several pesticide concentrations were higher than the average pesticide in the rest of the brook trout but were not higher than the maximum concentrations among other brook trout. Concentration differences are typically less than a factor of 4. It would be impossible to draw strong conclusions with data from only one hatchery fish.

5.4 Division of Hazard Index by Target Organ

For receptors with total hazard indices greater than one, we divided the total HI for direct exposure pathways (i.e. ingestion and dermal contact) by primary target organ. Table 16 below shows the target organ categories used in the HI division for the ingestion and dermal exposure pathways only. We chose not to divide HIs for the inhalation route of exposure because these HIs are based on modeled rather than measured EPCs.

Table 16. Division of COPCs by Primary Target Organ

Compound of Potential Concern (COPC)	Primary Target Organ (RfD) from IRIS File	Target Organ Category for HI Division
Volatile Organic Compounds		
Benzene	bone marrow	hematologic
n-Butylbenzene	NA	-
sec-Butylbenzene	NA	-
tert-Butylbenzene	NA	-
Chloroethane	fetus	reproductive
Chloromethane	central nervous system	central nervous
1,2-Dichlorobenzene	no observed effect	attributed to all
Dichlorodifluoromethane	reduced body weight	attributed to all
1,1-Dichloroethane	no observed effect	attributed to all
1,2-Dichloroethane	NA	-
1,1-Dichloroethene	liver	liver
1,2-Dichloroethene (trans)	increased serum alkaline phosphatase in male mice	liver
1,2-Dichloroethene (cis)	assume same as trans-1,2-DCE	liver
Ethylbenzene	liver and kidney toxicity	liver, kidney
para-Isopropyltoluene	NA	-
Methylene Chloride	liver	liver
Methyl-tert-butyl ether	NA	-
n-Propylbenzene	NA	-
1,1,2,2-Tetrachloroethane	NA	-
Tetrachloroethene	hepatotoxicity in mice, weight gain in rats	liver
Toluene	changes in liver and kidney weights	liver, kidney
1,1,1-Trichloroethane	central nervous system	central nervous
Trichloroethene	NA	-
1,2,4-Trimethylbenzene	NA	-
1,3,5-Trimethylbenzene	NA	-
Vinyl Chloride	liver cell polymorphism	liver
Petroleum Fractions		
Volatile Petroleum Hydrocarbons (VPH)		
C5-C8 aliphatics	liver , kidney	liver, kidney
C9-C12 aliphatics	liver , hematological	liver, hematologic
C9-C10 aromatics	decreased body weight	attributed to all
Extractable Petroleum Hydrocarbons (EPH)		
C9-C18 aliphatics	liver , hematological	liver, hematologic
C11-C22 aromatics	decreased body weight	attributed to all
Semivolatile Organic Compounds (SVOCs)		
Non-Carcinogenic PAHs		
Acenaphthylene	kidney	kidney
Benzo(ghi)perylene	kidney	kidney
2-Methylnaphthalene	decreased body weight	attributed to all
Naphthalene	decreased body weight	attributed to all
Phenanthrene	kidney	kidney
Carcinogenic PAHs		
Benz(a)anthracene	NA	-
Benzo(a)pyrene	NA	-
Benzo(b)fluoranthene	NA	-
Benzo(k)fluoranthene	NA	-
Chrysene	NA	-

Table 16. Division of COPCs by Primary Target Organ

Compound of Potential Concern (COPC)	Primary Target Organ (RfD) from IRIS File	Target Organ Category for HI Division
Dibenzo(ah)anthracene	NA	-
Indeno(123-cd)pyrene	NA	-
Phthalates		
bis(2-Ethylhexyl)phthalate	liver (chronic)	liver
	testicular effects (subchronic)	reproductive
Pesticides		
Aldrin	liver toxicity	liver
alpha-HCH	NA	-
gamma-HCH (Lindane)	liver and kidney toxicity	liver, kidney
4,4'-DDE	assume same as 4,4'-DDT	liver
4,4'-DDT	liver lesions	liver
Dieldrin	liver lesions	liver
Heptachlor	liver weight increases in males	liver
Heptachlor Epoxide	increased liver-to-body weight ratio in both sexes	liver
Trans-nonachlor	NA	-
Cis-nonachlor	NA	-
Polychlorinated Biphenyls (Aroclor 1254)	ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toenails, decreased antibody (IgG and IgM) response to sheep erythrocytes	immune
Inorganic Compounds		
Antimony	longevity, blood glucose, cholesterol	attributed to all
Arsenic (inorganic)	hyperpigmentation, keratosis and possible vascular complications	skin, circulatory
Barium	increased kidney weight	kidney
Beryllium	small intestinal lesions	gastrointestinal
Cadmium (Food)	significant proteinuria	kidney
Cadmium (Water)	significant proteinuria	kidney
Chromium (VI)	none reported	attributed to all
Copper	gastrointestinal tract	gastrointestinal
Manganese	central nervous system effects	central nervous
Mercury (methyl)	central nervous system	central nervous
Molybdenum	increased uric acid levels	kidney
Nickel (soluble salts)	decreased body and organ weights	attributed to all
Selenium	clinical selenosis	attributed to all
Thallium	liver	liver
Vanadium	decreased hair cystine	attributed to all
Zinc	47% decrease in erythrocyte superoxide dismutase(ESOD) concentration in adult females after 10 weeks of zinc exposure	attributed to all
Nitrate-N	early clinical signs of methemoglobinemia in excess of 10% (0-3 months old infant formula)	circulatory

Notes:

(1) NA - Not available

(2) Compounds associated with "no observed effect," "decreased body weight," and "decreased organ weight" are assumed to affect all target organ categories.

(3) Target organ categories: hematologic, reproductive, central nervous [system], kidney, liver, immune, gastrointestinal, and skin

Ideally, this division would be performed by mechanism of toxicity, accounting for toxic outcomes (and consequently target organs, tissues) that might occur at estimated exposure duration, frequency, and magnitude. However, the number of COPCs and limited toxicity literature preclude such an in depth analysis to support division of HIs.

5.4.1 Future Resident

A detailed summary of risk and hazard estimates for all COPCs for the future resident are presented Tables 9.1 through 9.4 in Appendix P and Tables 10.1 through 10.4 in Appendix Q. Total hazard indices for the RME and CT cases exceeded one; therefore, we divided the HIs into target organ categories. The target organ categories include: hematologic, reproductive, central nervous, kidney, circulatory, immune, gastrointestinal, skin, and liver.

Individual target organ HIs still exceed one for all nine of the target organ categories for the future child resident (0-1 ft and 0-10 ft soil EPC, RME and CT) and for the future adult resident (0-1 ft and 0-10 ft soil EPC, RME case only). The CT hazard indices for the future adult resident (both soil EPCs) exceed one for the following categories: hematologic, liver, circulatory, central nervous, immune, and skin.

When considering the HI from risk driver COPCs only, the target organ HIs exceed one for the hematologic, central nervous, kidney, circulatory, immune, skin, and liver target organ categories for the future adult resident and future child resident RME scenario (both soil EPCs). Individual target organ HIs exceed one for the immune and central nervous target organ categories for the Adult CT scenarios (both soil EPCs). For the Child CT scenario (both soil EPCs), the target organ HIs exceed one for central nervous, circulatory, immune and skin categories.

5.4.2 Current Resident

The hazard indices for the current resident were not greater than one; therefore, we did not divide the total hazard index into target organ categories.

5.4.3 Future Construction Worker

A detailed summary of risk and hazard estimates for all COPCs for the future construction worker is presented in Table 9.10 in Appendix P and Table 10.10 in Appendix Q. The division of the total HI by target organ is also shown on these tables.

Individual target organ HIs still exceed one for all nine of the target organ categories for the RME case. For the CT case, the HIs for the hematologic, central nervous, and immune target organs exceed one. When considering the HI from risk driver COPCs only, the target organ HIs exceed one for the hematologic, central nervous, kidney, immune and liver target organs in the RME case. Only the immune HI exceeds one in the CT case, which is simply the HI for PCBs.

5.4.4 Current Trespasser

A detailed summary of risk and hazard estimates for all COPCs for the current trespasser are presented in Table 9.9 in Appendix P and Tables 10.9 in Appendix Q. The division of the total HI by target organ is also shown on these tables. The HI for the immune target organ category exceeds one in the RME and CT cases for the current trespasser. The immune HI is largely attributable to the hazard from PCBs.

5.4.5 Current/Future Recreational Person

A detailed summary of risk and hazard estimates for all COPCs for the recreational person is presented in Table 9.8 in Appendix P and Table 10.8 in Appendix Q. The division of the total HI by target organ is also shown on these tables. The HI for the central nervous and immune target organ categories exceeds one in the RME case. The HI for PCBs contributes most to the immune target organ HI. The central nervous target organ HI is largely due to the HI for manganese. The total HI does not exceed one for the CT case.

5.4.6 Current/Future Fisherperson

A detailed summary of risk and hazard estimates for all COPCs for the fisherperson is presented in Table 9.7 in Appendix P and Table 10.7 in Appendix Q. The division of the total HI by target organ is also shown on these tables. The HI for the immune target organ category exceeds one in the RME case. The HI for PCBs contributes most to the immune target organ HI. The total HI does not exceed one for the CT case.

6.0 UNCERTAINTY ANALYSIS

The non-cancer hazard and cancer risk estimates in this assessment are subject to numerous uncertainties. In each step of this assessment, we addressed uncertainty by making assumptions that would overestimate rather than underestimate risk. Consequently, non-cancer hazard and cancer risk estimates likely overestimate the actual risk associated with exposures to COPCs at the site. Because we did not use probabilistic analysis to estimate non-cancer hazard and cancer risk, we cannot estimate the level of confidence in the point estimates for each human receptor.

The following sections summarize sources of uncertainty in each of the four risk assessment steps. Rather than providing an exhaustive list of sources of uncertainty, each section describes key sources for that step in the risk assessment.

6.1 Hazard Identification

The hazard identification is subject to uncertainties related to limitations of sampling design and collection and analytical techniques. These uncertainties are discussed in greater detail in Data Useability Worksheets found in Appendix B. This section summarizes some of the more important sources of uncertainty and their implications for interpreting risk estimates for the Site.

6.1.1 Data Quality Issues

COPC detection limits were elevated in soil samples with high levels of PCBs and/or petroleum contamination. In most cases, this source of uncertainty lead to conservative risk estimates where ½ these detection limits were used. However, some VOCs in soil might have been screened out due to infrequent detection at these higher detection limits. However, where lower detection limits were attained, VOCs were typically not detected in soil. In any event, VOC contamination in soil must be addressed with remediation to remove this source of contamination to groundwater at the Site, particularly in areas such as the former lagoon and waste oil UST.

The risk assessment used Aroclor data as well as homolog and congener data. The Aroclor data are somewhat low biased due to:

- Low recoveries combined with external calibration;
- Differential weathering of individual peaks in the PCB pattern; and
- Non-inclusion of PCB peaks that do not match the Aroclor pattern (O'Reilly, Talbot & Okun, Sept 2000).

Despite the low bias, this risk assessment uses Aroclor data because its exclusion would result in the exclusion of some of the highest PCB concentrations measured at the Site. Even with the low bias, PCB cancer risks of concern have been estimated for a number of exposure scenarios.

Pesticide data for all Site media are questionable given analytical interferences. Where false positives were possible, we assumed the pesticides were present for the purpose of estimating risk. Consequently, risk estimates for pesticides should be interpreted and used with caution.

6.1.2 Lack of Toxicity Criteria

Not all COPCs have toxicity values to quantify non-cancer hazard and cancer risk associated with human exposure to them. For example, risk from MTBE in groundwater could not be quantified in this assessment due to a lack of toxicity information. Therefore, as discussed in Section 4.4, MTBE levels in groundwater might represent levels of concern and should be addressed along with VOCs in any remediation plan. MTBE is found in Site groundwater; however, it is likely from off-site source(s) according to the preliminary draft RI (SHA, 1999).

6.2 Exposure Assessment

The exposure assessment is subject to uncertainties associated with sampling, analysis, and limitations in attaining data that best reflect the concentrations to which people are likely to be exposed. This section highlights some of the more important sources of exposure assessment uncertainty and their implications for interpreting risk estimates for the Site.

6.2.1 Uncertainty in Average Daily Intake Calculations

We cannot provide a quantitative assessment of confidence in average daily intake estimates without using distribution analyses. Given the large effort required to conduct such analyses, we instead provide CT and RME intake calculations to provide a range of possible risks at the Site.

Scientific understanding and regulatory guidance continue to evolve regarding dermal adherence and dermal absorption for both soil and water exposures. This assessment employs reasonable CT and RME estimates for these values based on the most recent literature.

Exposure is estimated for some exposure pathways using screening models (i.e. fugitive dust and soil vapor inhalation, tap water inhalation). Non-cancer hazard and cancer risk estimates for these modeled exposure pathways should be viewed as conservative screening values only.

Risk estimates from consumption of brook trout fillets from Kelley Brook fish do not account for COPC loss during cooking. For example, such loss can occur when lipophilic COPCs partition to oil or butter that fish is cooked in and then discarded.

While all RME exposure factors are selected to represent realistic maximum exposures, the soil ingestion rate of 480 mg/d for the future construction worker has been questioned. In recent work with non-smoking, non-geophagic adults, Kissel et al. (1998) concluded that an ingestion rate of 480 mg/d appears to be implausible. In this study, adult volunteers reported that 10 mg of soil in the mouth was readily detected and unpleasant. Therefore, it is unlikely that repeated, unintentional ingestion of that mass of soil would occur to reach a 480 mg/d ingestion rate.

6.2.2 Home Garden Exposure Pathway

Exposure to Site contaminants from a home garden was not quantified. Some organic compounds (e.g., PCBs and PAHs) and metals can be taken up by plants to varying degrees. Inclusion of this exposure pathway could result in higher risk estimates for the future resident. Estimated risk to a future resident already exceeds USEPA and New Hampshire risk criteria without quantifying COPC exposure from a home garden.

6.2.3 Vapor Intrusion Exposure Pathway for a Future Resident

As discussed in Section 5.3.1, a future resident might be exposed to groundwater or soil COPCs that migrate into future homes via soil gas. However, this exposure would likely contribute minimally relative to exposure from ingesting and bathing in contaminated tap water. For this reason along with the uncertainty associated with modeling soil gas vapor intrusion for hypothetical buildings, this exposure pathway was not quantified. It will be important to quantify this exposure pathway if homes are built on the Site, without remediation of groundwater. Also, there are some areas of VOC contamination of soil (e.g., the lagoon) that would be a concern for construction of future homes.

6.3 Dose-Response Assessment

Major sources of uncertainty concerning the toxicity assessment include the extrapolation from high doses in animals to low doses in humans for non-carcinogens and carcinogens, and conservative assumptions built into derivation of RfDs, RfCs, and CSFs.

6.3.1 Uncertainty in the RfDs and RfCs

For example, RfDs and RfCs might incorporate uncertainty factors to address the following sources of uncertainty:

- the expected differences in responsiveness between humans and animals
- variability among individuals within the human population
- extrapolation from a LOAEL to a NOAEL
- extrapolation from a subchronic to chronic exposure
- an inadequate toxicity data base

6.3.2 PCB Cancer Risk Estimates

USEPA recently revised its approach for quantifying PCB cancer risk, which accounts for differences in PCB congener mixtures found in various environmental media. This approach also calls for quantification of dioxin-like PCB cancer risk in addition to PCB cancer risk. However, adding dioxin-like PCB congener cancer risk and PCB cancer risk probably involves some amount of “double-counting” because the dioxin-like congeners were present in the PCB test material used in the toxicity study used to derive the PCB cancer slope factor.

6.3.3 Petroleum Hazard Indices

There is a great deal of uncertainty associated with petroleum toxicity. Because toxicity information is so limited, a fraction-based approach is used that is designed to provide a conservative representation of potential human health toxicity.

6.4 Risk Characterization

Sources of uncertainty throughout the risk assessment affect risks and non-cancer hazards estimated in the Risk Characterization. In addition, more sources of uncertainty are introduced in deciding how to sum cancer risks and non-cancer hazards across COPCs and exposure pathways. In this assessment, we added

cancer risks and non-cancer hazards as appropriate to represent reasonable cumulative exposures now and in the future.

6.4.1 Combined Child and Adult Future Resident Cancer Risk

In the RAGS Part D tables and in this assessment, potential cancer risk is reported separately for the future child and adult resident. However, these estimates should be combined for a future resident who spends the first thirty years of life living at the Site. The combined risk estimates for a future resident is shown in section 5.3.1 of the text.

6.4.2 Division of Hazard Indices

Non-cancer dermal and ingestion exposure pathway HIs for each receptor were divided by primary target organ. However, there is a great deal of uncertainty associated with this division for a number of reasons:

- COPCs may target multiple organs
- Relationships between dose level and adverse effects can be complex
- It is extremely difficult to judge whether the complex mixtures present at the Site might act additively, synergistically, or antagonistically

6.4.3 VOC Concentration Trends in Neighboring Private Wells

VOC concentrations are increasing in some wells near the Site. It is important that NHDES continue to monitor these wells and ensure that cumulative exposure to contamination in well water does not pose risk in excess of USEPA and New Hampshire risk criteria.

7.0 RECOMMENDATIONS FOR PRELIMINARY REMEDIATION GOALS

In consultation with USEPA, NHDES, NHDHHS, and SHA, MCA developed preliminary remediation goals (PRGs) for a subset of COPCs for soil and groundwater (Table 17). During these consultations, recommendations were made and adopted for modifying PRGs. All modifications were intended to provide PRGs that are both technically attainable and in compliance with applicable federal and state regulations and policy. The approach used to derive the PRGs is described in detail elsewhere (MCA 2000).

Several "non-risk driver" COPCs do not have PRGs. These include dioxin congeners and several VOCs and metals.

Dioxin congeners represent a set of COPCs for which no PRGs were assigned. Dioxin risk at the Site is primarily due to dioxin-like PCB congeners, not dioxin congeners, even though soil samples were collected from where dioxin concentrations were expected to be highest. Dioxin and PCB contamination are likely to be detected in the same locations; therefore, dioxin congeners will be remediated when PCBs are remediated. Furthermore, dioxin congeners alone contribute 0.09 ppb of TEQ, while PCBs contribute about 5 ppb TEQ. Consequently, no PRGs are established for dioxin congeners.

Other COPCs without PRGs include alkylbenzene COPCs in soil and groundwater, but they are represented in the petroleum fraction PRGs. Nine metals (antimony, barium, beryllium, vanadium, zinc, molybdenum, cadmium, copper, and manganese) and two VOCs (cis-1,2-DCE and tetrachloroethylene) in soil do not have PRGs; however, these compounds are almost never detected above Region III RBCs. Two metals in groundwater (barium and selenium) do not have PRGs.

The primary reason for not assigning PRGs to these compounds is that they make a very small contribution to non-cancer hazard and cancer risk estimates at the Site. Moreover, they typically are coincident with other COPCs that do have PRGs assigned to them. Therefore, any remediation strategy that addresses COPCs with PRGs will likely address these other COPCs.

We identified significant risk associated with human exposure to Kelley Brook sediment and surface water near the historic discharge area as well as from consumption of brook trout. However, no human health-based PRGs have been recommended for these media. The draft baseline Ecological Risk Assessment for this Site discusses the need to attain sediment concentrations of COPCs in the discharge area that are consistent with those that have been measured in upstream and downstream sampling locations. If this is accomplished, human health risk associated with recreational and fishing activities in Kelley Brook should be reduced.

Table 17. COPCs for which the individual cancer risk exceeds 1E-6 and/or the hazard quotient is greater than 1. PRGs were developed for these COPCs.

Soil	Groundwater
C11-C22 Aromatics	Benzene
Benzo(a)pyrene RPQ	1,1-Dichloroethane
bis(2-Ethylhexyl)phthalate	1,2-Dichloroethane
Polychlorinated Biphenyls	1,1-Dichloroethene
Arsenic	1,2-Dichloroethene (cis)
Chromium VI (particulates)	Ethylbenzene
Lead	Methylene Chloride
Mercury	Toluene
Nickel	1,1,1-Trichloroethane
	1,1,2,2-Tetrachloroethane
	Tetrachloroethene
	Trichloroethene
	Vinyl Chloride
	C9-C10 Aromatics
	C11-C22 Aromatics
	Naphthalene
	Aldrin
	alpha-BHC
	gamma-BHC (Lindane)
	Dieldrin
	Heptachlor
	Heptachlor Epoxide
	Antimony
	Arsenic
	Cadmium (Water)
	Chromium VI (aerosols)
	Manganese

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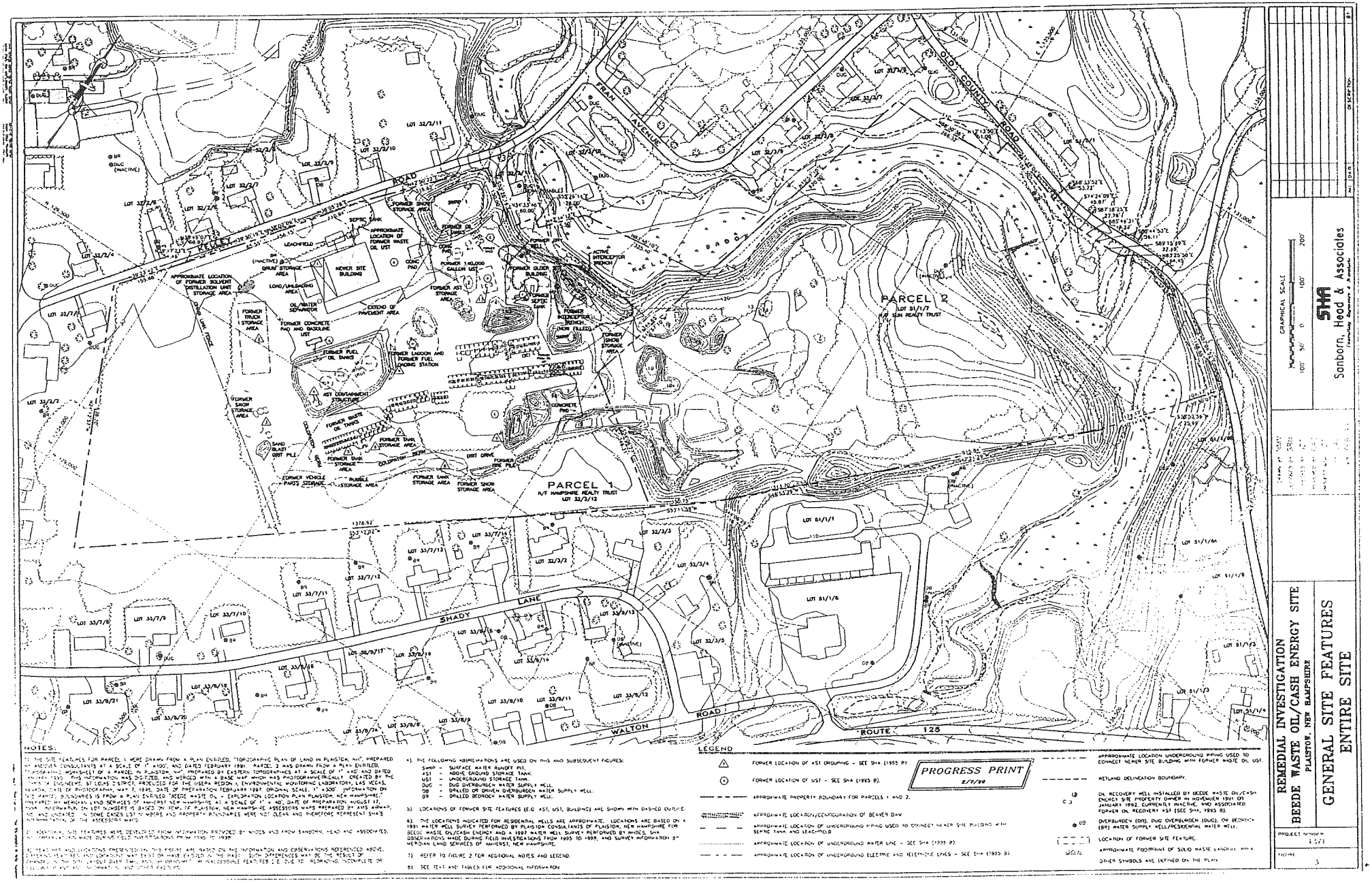
FIGURES

Figure 1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DIXOT COUNTY ROAD
SHADY LANE
WALTON ROAD
ROUTE 125
PROGRESS PRINT
2/7/89
REMEDIAL INVESTIGATION
BEEDE WASTE OIL/CASH ENERGY SITE
PLAISTOW, NEW HAMPSHIRE
GENERAL SITE FEATURES
ENTIRE SITE
Sanborn, Head & Associates
GRAPHICAL SCALE
1" = 100'
PROJECT NUMBER
1571
FIGURE
1

FIGURE 2
Map of Kelley Brook Reaches
Plaistow, New Hampshire

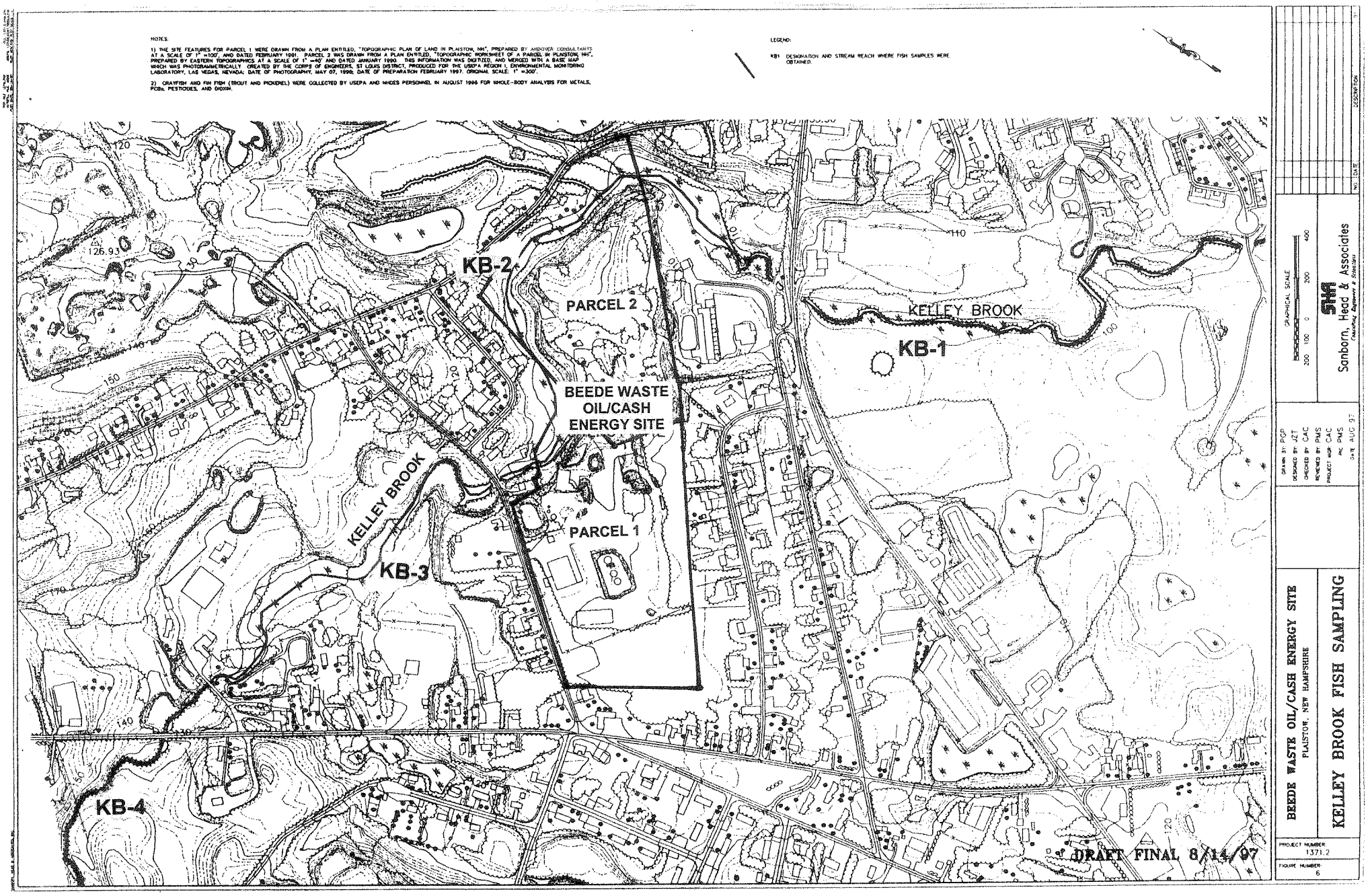


Figure 3. Total Metal Concentrations in Fish and Shellfish
Kelley Brook, Plaistow, New Hampshire

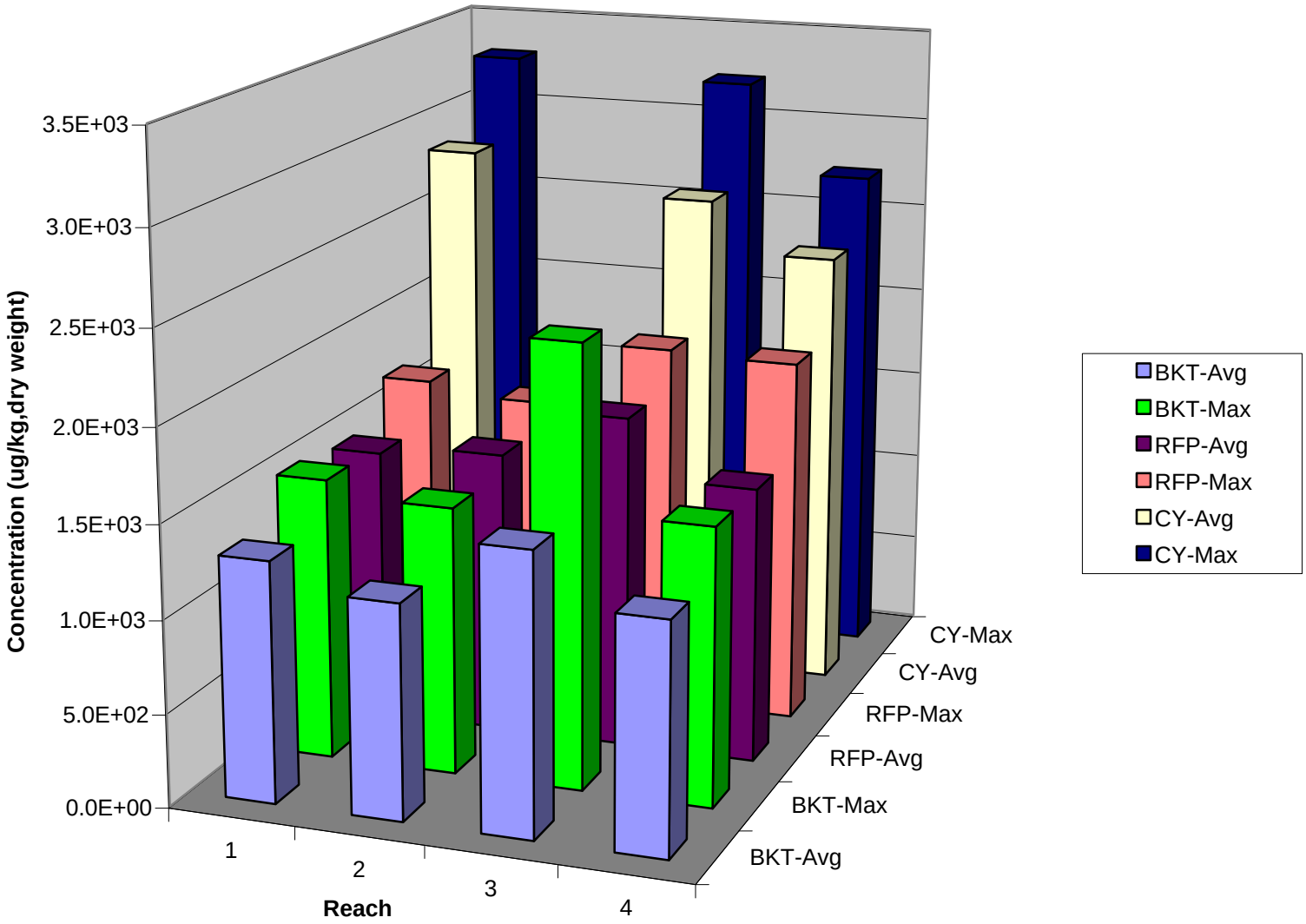
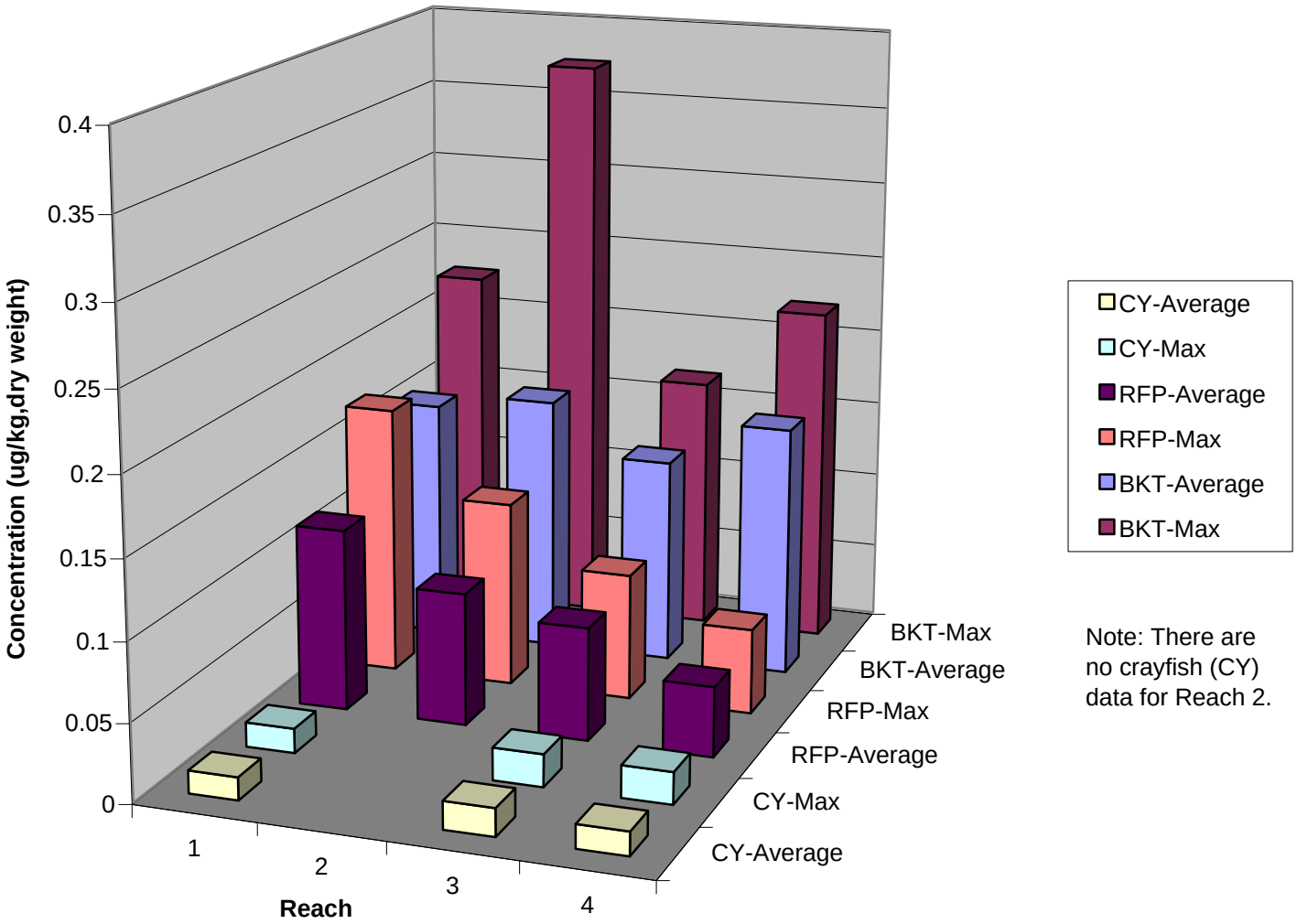
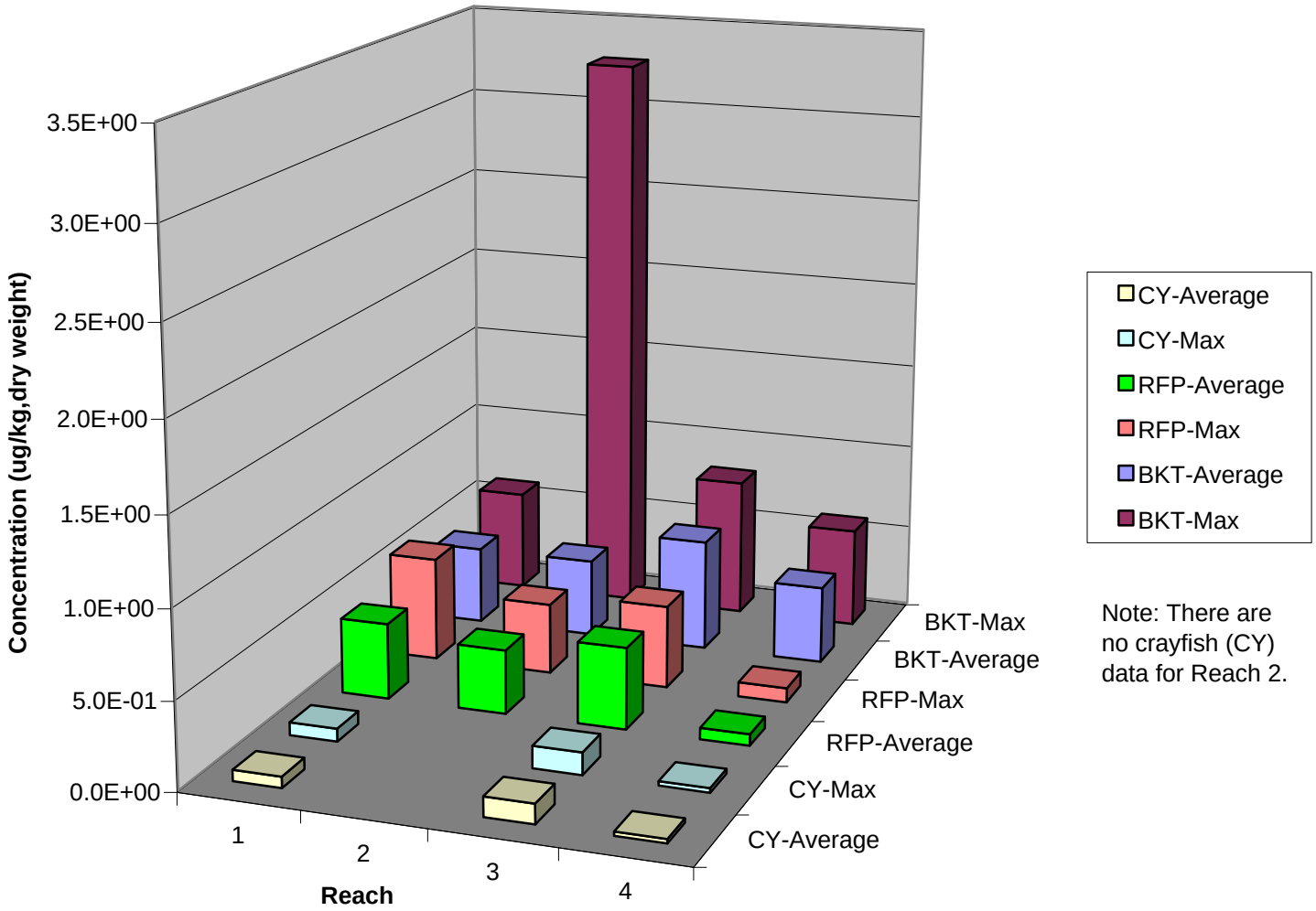


Figure 4. Total Pesticide Concentrations in Fish and Crayfish
Kelley Brook, Plaistow, New Hampshire



Note: There are no crayfish (CY) data for Reach 2.

Figure 5. Total PCB Concentrations in Fish and Crayfish
Kelley Brook, Plaistow, New Hampshire



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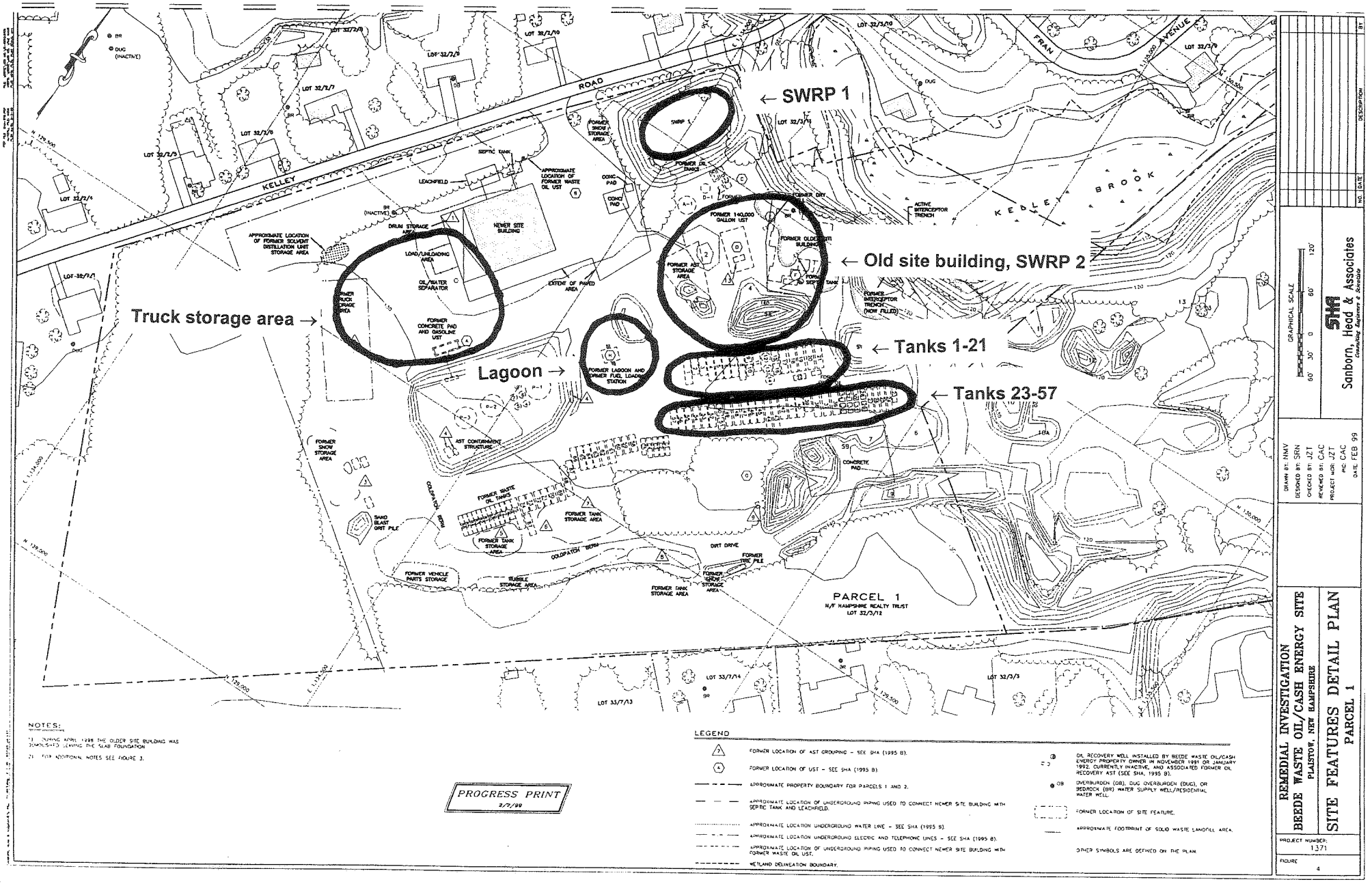
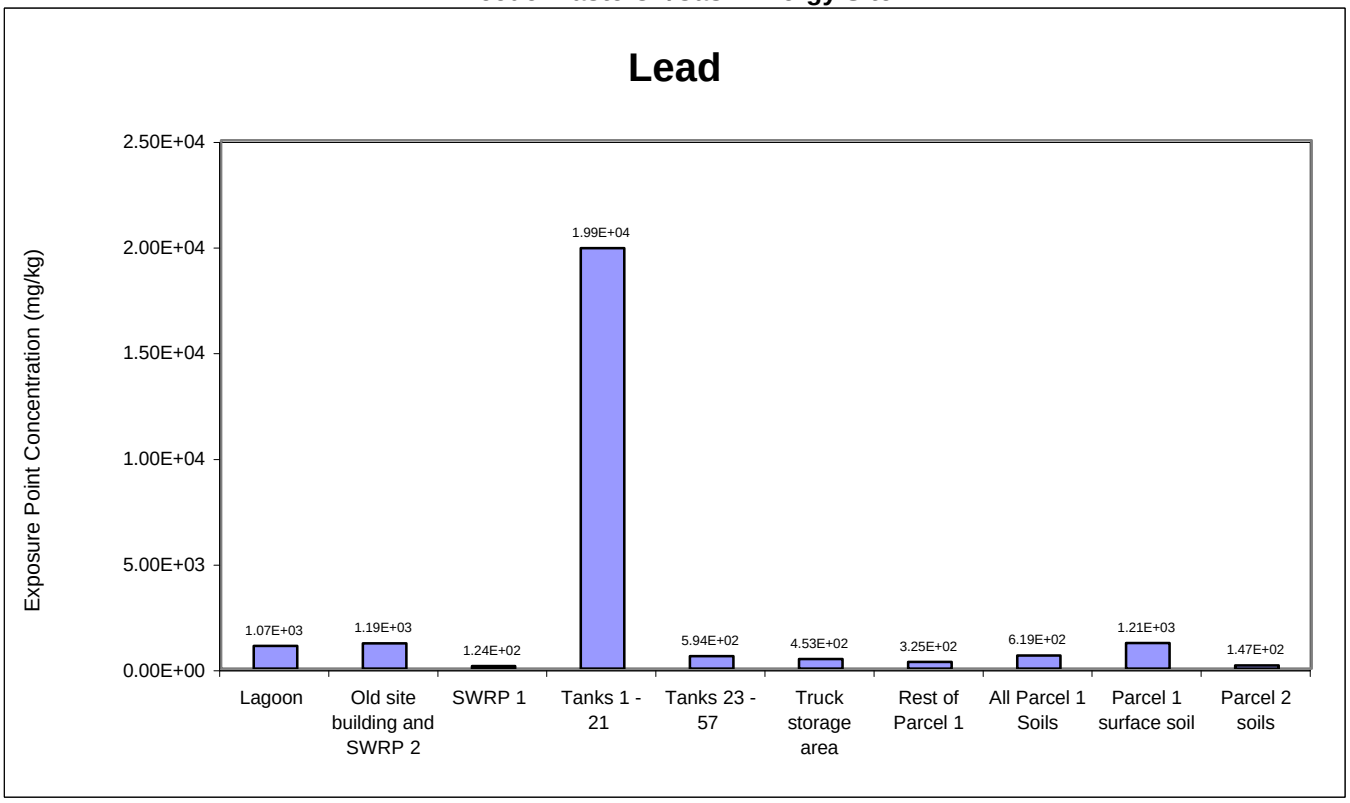


Figure 6. Potential Soil Exposure Areas

Figure 7
Exposure Point Concentrations for Lead and PCBs in Ten Potential Soil Exposure Areas
Beede Waste Oil/Cash Energy Site



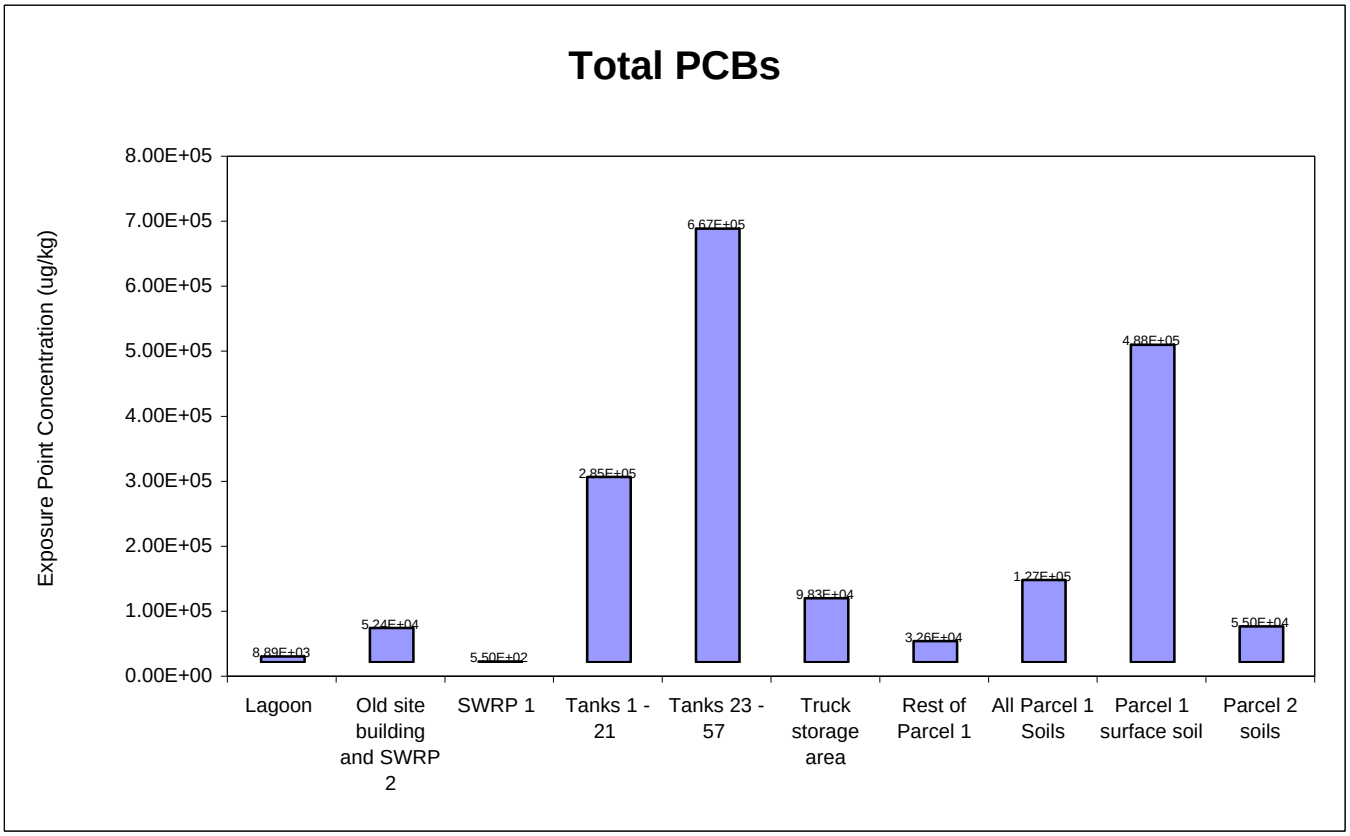
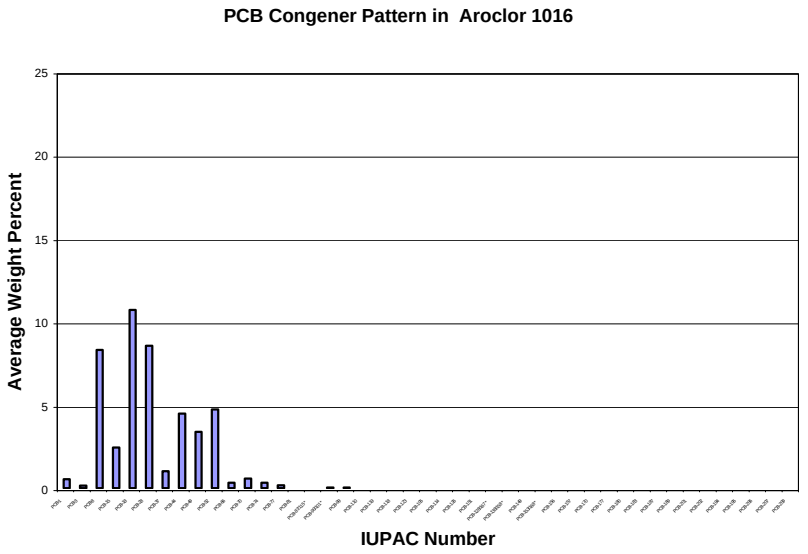
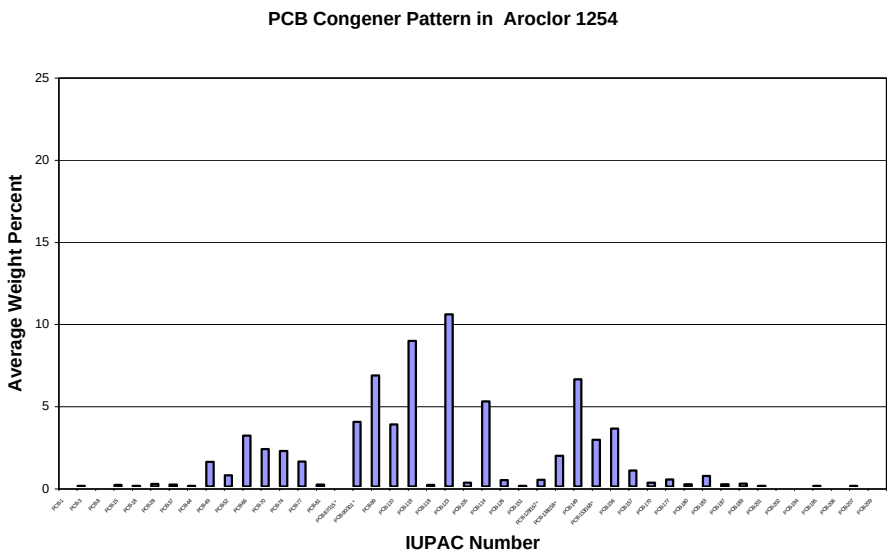
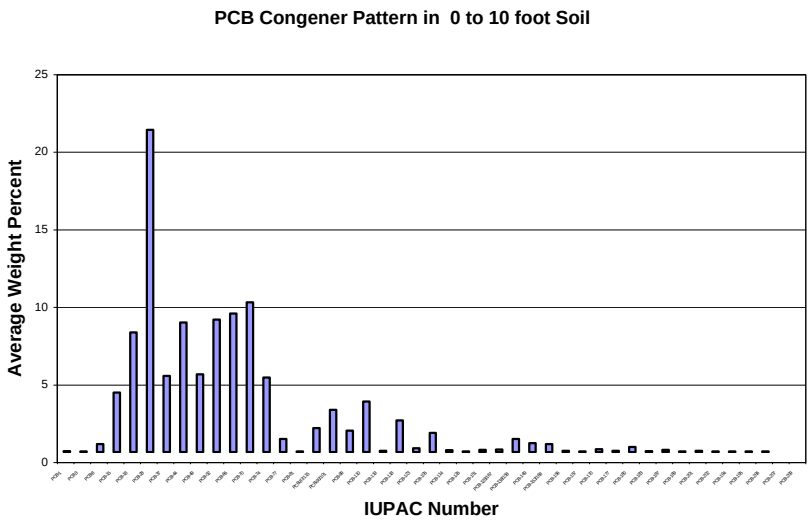
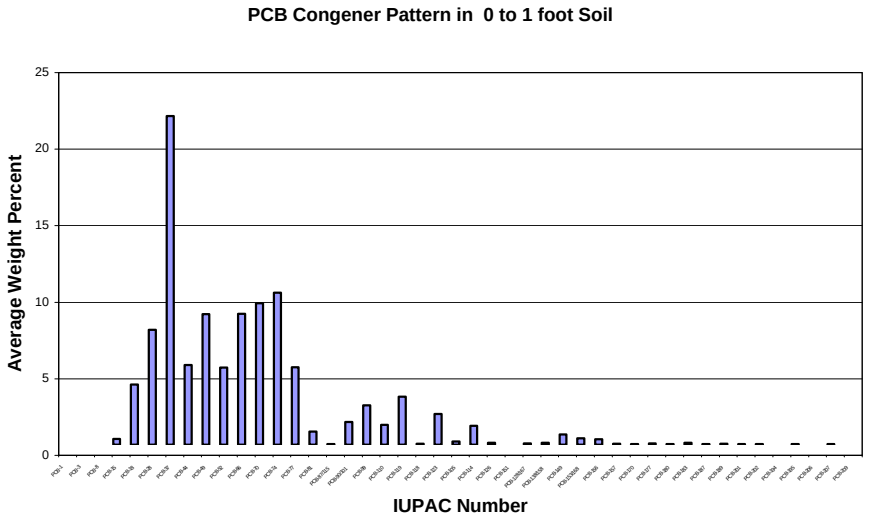


Figure 8. Comparison of PCB Congener Patterns in Site Soils with Aroclor 1016 and Aroclor 1254



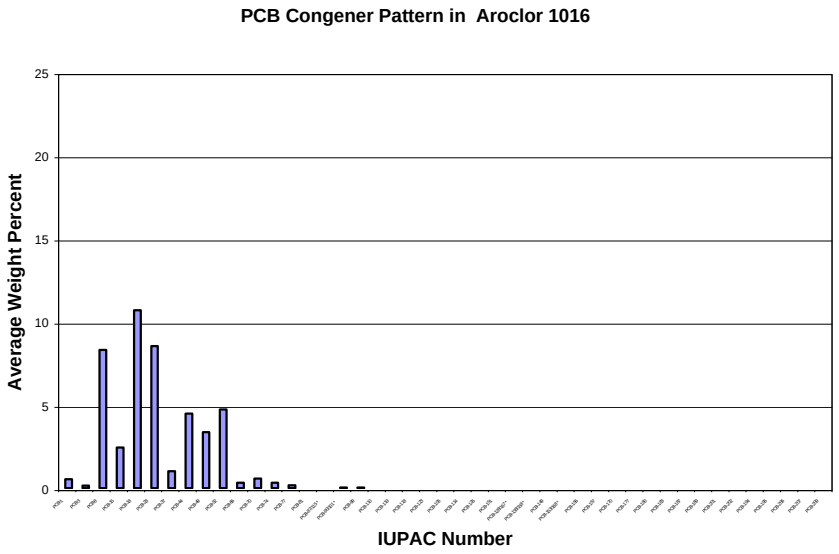


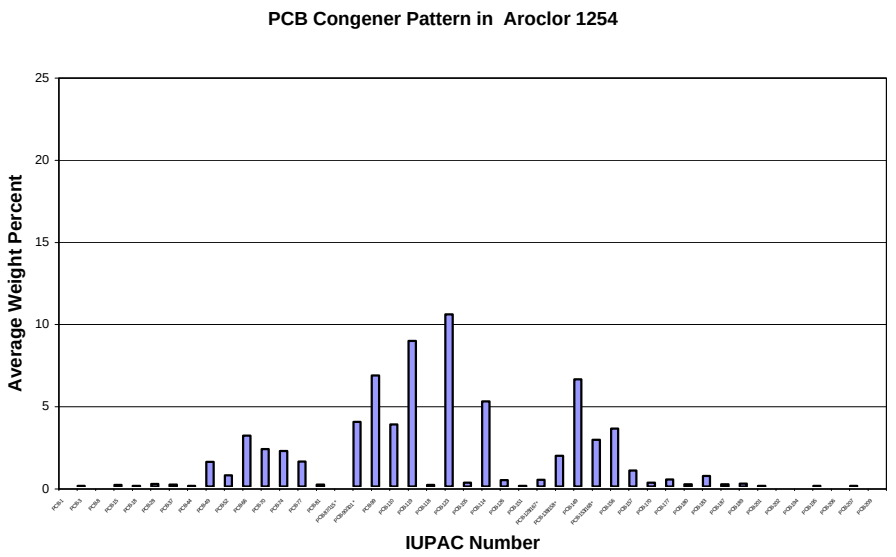


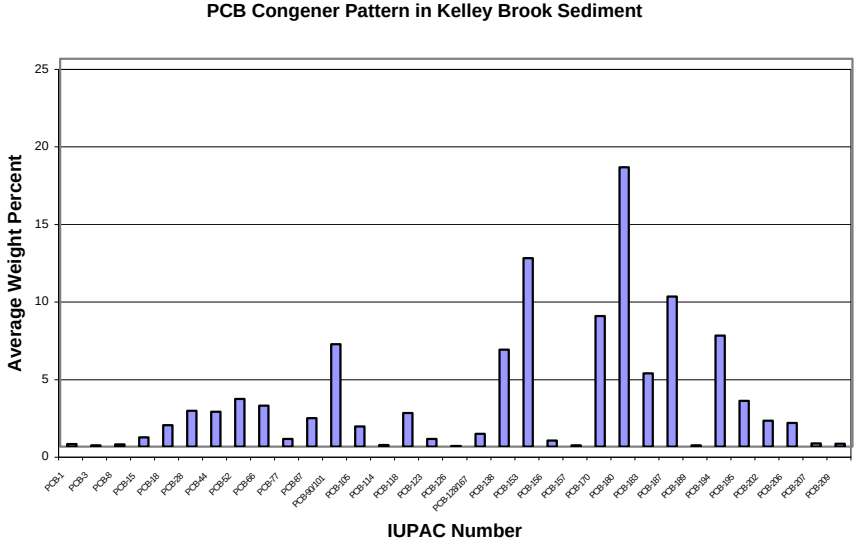


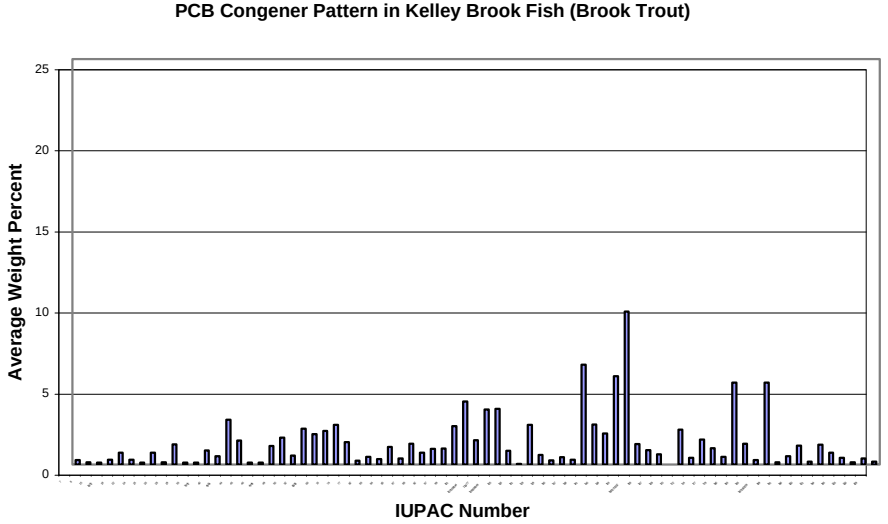
Note: Aroclor congener patterns were produced using data from Frame et al. 1996.

Figure 9. Comprison of PCB Congener Patterns in Kelley Brook Sediment and Fish with Aroclor 1016 and Aroclor 1254









Note: Aroclor congener patterns were produced using data from Frame et al. 1996.